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NEWS 19 Jan 29 Simultaneous left and right truncation added to COMPENDEX, ENERGY, INSPEC
NEWS 20 Feb 13 CANCERLIT is no longer being updated
NEWS 21 Feb 24 METADEX enhancements
NEWS 22 Feb 24 PCTGEN now available on STN
NEWS 23 Feb 24 TEMA now available on STN
NEWS 24 Feb 26 NTIS now allows simultaneous left and right truncation
NEWS 25 Feb 26 PCTFULL now contains images
NEWS 26 Mar 04 SDI PACKAGE for monthly delivery of multifile SDI results
NEWS 27 Mar 19 APOLLIT offering free connect time in April 2003
NEWS 28 Mar 20 EVENTLINE will be removed from STN
NEWS 29 Mar 24 PATDPAFULL now available on STN
NEWS 30 Mar 24 Additional information for trade-named substances without structures available in REGISTRY
NEWS 31 Mar 24 Indexing from 1957 to 1966 added to records in CA/CAPLUS
NEWS 32 Apr 11 Display formats in DGENE enhanced
NEWS 33 Apr 14 MEDLINE Reload

NEWS EXPRESS April 4 CURRENT WINDOWS VERSION IS V6.01a, CURRENT MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP), AND CURRENT DISCOVER FILE IS DATED 01 APRIL 2003
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FILE 'HOME' ENTERED AT 14:06:11 ON 16 APR 2003

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'REGISTRY' ENTERED AT 14:06:16 ON 16 APR 2003

Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 15 APR 2003 HIGHEST RN 503084-53-5
DICTIONARY FILE UPDATES: 15 APR 2003 HIGHEST RN 503084-53-5

TSCA INFORMATION NOW CURRENT THROUGH MAY 20, 2002

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Experimental and calculated property data are now available. See HELP PROPERTIES for more information. See STNote 27, Searching Properties in the CAS Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

=> e ephrin b2

E1	1		EPHRIDINE/BI
E2	170		EPHRIN/BI
E3	0	-->	EPHRIN B2/BI
E4	42		EPHRINE/BI
E5	2		EPHROITE/BI
E6	1		EPHRONE/BI
E7	3		EPHROS/BI
E8	1		EPHTHAL/BI
E9	1		EPHTHALEXON/BI
E10	1		EPHTHALIC/BI
E11	3		EPHTHLA054C07/BI
E12	7		EPHX1/BI

=> s e2

L1	170	EPHRIN/BI

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	4.62	4.83

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FILE 'EMBASE' ENTERED AT 14:06:39 ON 16 APR 2003
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=> s l1
L2          144 L1

=> s l2 and (tumor? or tumour?)
L3           52 L2 AND (TUMOR? OR TUMOUR?)

=> s l2 and vascul?
L4           33 L2 AND VASCUL?

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5            33 DUP REM L4 (0 DUPLICATES REMOVED)

=> s l3 not l4
L6           37 L3 NOT L4

=> s l5 or l6
L7           70 L5 OR L6

=> dup rem l7
PROCESSING COMPLETED FOR L7
L8            67 DUP REM L7 (3 DUPLICATES REMOVED)

=> d ibib ab 1-
YOU HAVE REQUESTED DATA FROM 67 ANSWERS - CONTINUE? Y/(N):y
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L8 ANSWER 1 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2003:270224 CAPLUS
TITLE: Gene expression profiles useful in methods of diagnosis of cancer compositions and methods of screening for modulators of cancer
INVENTOR(S): Afar, Daniel; Aziz, Natasha; Gish, Kurt C.; Hevezi, Peter A.; Mack, David H.; Wilson, Keith E.; Zlotnik, Albert
PATENT ASSIGNEE(S): Eos Biotechnolgy, Inc., USA
SOURCE: PCT Int. Appl., 767 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 21
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003025138	A2	20030327	WO 2002-XE29560	20020917
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2003025138	A2	20030327	WO 2002-US29560	20020917
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.:
US 2001-323469P P 20010917
US 2001-323887P P 20010920
US 2001-350666P P 20011113
US 2002-355145P P 20020208
US 2002-355257P P 20020208
US 2002-372246P P 20020412
WO 2002-US29560 A 20020917

AB Described herein are genes whose expression are up-regulated or down-regulated in specific cancers, including acute lymphocytic leukemia, glioblastoma, glioblastoma multiforme, glioma, kidney cancer, stomach cancer, melanoma, and benign NEVI. Mol. profiles of various normal and cancerous tissues were detd. and analyzed using the Affymetrix/Eos Hu01 and Hu03 GeneChip microarrays contg. 35,403 and 59,680 probe sets, resp. Related methods and compns. that can be used for diagnosis and treatment of those cancers are disclosed. Also described herein are methods that

L8 ANSWER 1 OF 67 CAPLUS COPYRIGHT 2003 ACS (Continued)
can be used to identify modulators of selected cancers. [This abstr. record is one of nine records for this documents necessitated by the large
no. of index entries required to fully index the document and publication system constraints.).

L8 ANSWER 2 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2003:202620 CAPLUS
DOCUMENT NUMBER: 138:238156
TITLE: Preparation of benzofuro[3,2-c]quinolines and related compounds as protein tyrosine kinase inhibitors
INVENTOR(S): Eren, Doron; Zaliani, Andrea; Pe'er, David; Bogin, Oren; Yayon, Avner
PATENT ASSIGNEE(S): Prochon Biotech Ltd., Israel
SOURCE: PCT Int. Appl., 69 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003020698	A2	20030313	WO 2002-IL740	20020905
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.:
US 2001-317186P P 20010906
IL 2001-145329 A 20010909

AB Title heterocyclic compds. I and II [wherein X = N or O; R1 and R2 = independently halo, NO2, CN, CF3, alkyl, OR4, SR4, SOR5, SO2R5, CO2H, COR6, SONR7R8, SO2NR7R8, or NR7R8; R3 = H or R1 and is absent when X = O; R4 = H, alkyl, COR6, or CONR7R8; R5 = alkyl; R6 = H, alkyl, OR5, or NR7R8; R7 and R8 = independently H or alkyl; or one of R7 or R8 = H or alkyl, and the other = COR5, CO2R5, or CONR7R8; or NR7R8 = heterocyclyl; R9 and R10 = independently H or R1; m = 0-3 and n = 0-5 for I; m and n = independently 0-4 for II; with provisos; or stereoisomers or pharmaceutically acceptable salts thereof] were prepd. as protein tyrosine kinase inhibitors. For example, cyclization of di-Et 2-(2,4-dimethoxyphenyl)malonate and m-methoxyaniline in PhOPh gave 3-(2,4-dimethoxyphenyl)-4-hydroxy-7-methoxy-1H-quinolin-2-one (25%). Deprotection and cyclocondensation using pyridine.bul.HCl provided 3,9-dihydroxy-5H-benzofuro[3,2-c]quinolin-6-one (62.5%), which was converted to III (57.6%) by addn. of Me2NCOCl in the presence of TEA and dimethylaminopyridine in DMP. The latter inhibited fibroblast growth factor 9 (FGF9)-dependent proliferation of transfected FDCP-FR3 (R3) cells or FDCP-FR1 (R1) cells mediated by FGFR3 or FGFR1, resp., but did not affect interleukin 3 (IL-3)-dependent cell proliferation. In addn., III accelerated the growth of Ach369 femora by about 2 fold as compared with the untreated femora (4.33 vs. 2 units/day, resp.). Thus, I and II are useful for the treatment of proliferative, skeletal, and metabolic diseases and disorders assocd. with abnormal protein tyrosine kinase activity, including cancer and skeletal dysplasia.

L8 ANSWER 2 OF 67 CAPLUS COPYRIGHT 2003 ACS (Continued)

L8 ANSWER 3 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2003:97550 CAPLUS
DOCUMENT NUMBER: 138:164674
TITLE: Molecular markers for hepatocellular carcinoma and their use in diagnosis and therapy
INVENTOR(S): Debuschewitz, Sabine; Jobst, Juergen; Kaiser, Stephan
PATENT ASSIGNEE(S): Germany
SOURCE: PCT Int. Appl., 98 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003010336	A2	20030206	WO 2002-EP8305	20020725
DE 10136273	A1	20030213	DE 2001-10136273	20010725

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: DE 2001-10136273 A 20010725
AB The invention relates to mol. markers occurring for hepatocellular carcinoma. The invention more particularly comprises gene sequences or peptides coded thereby which can be regulated upwards or downwards for hepatic cell carcinoma (HCC) in relation to healthy, normal liver cells in the expression thereof. The invention also relates to the use of said sequences in the diagnosis and/or therapy of HCC and for screening purposes in order to identify novel active ingredients for HCC. The invention also relates to an HCC specific cluster as a unique diagnostic agent for HCC.

L8 ANSWER 4 OF 67 CAPLUS COPYRIGHT 2003 ACS (Continued)
prevention, diagnosis, characterization, and therapy of prostate cancer in a patient. Methods of treating prostate cancer are also provided. Compns., kits, and methods for detecting, characterizing, preventing, and treating human prostate cancers are provided.

L8 ANSWER 4 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2003:97279 CAPLUS
DOCUMENT NUMBER: 138:132255
TITLE: Differentially expressed gene gene and protein markers for identification, assessment, prevention, and therapy of prostate cancer
INVENTOR(S): Schlegel, Robert; Monahan, John E.; Endege, Wilson O.; Gannavarapu, Manjula; Gorbatcheva, Bella; Hoersh, Sebastian; Kamatkar, Shubhangi; Wonsey, Angela M.; Glatt, Karen; Zhao, Xumei; Anderson, Dustin
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 99 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003009814	A2	20030206	WO 2002-US23913	20020725

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:
US 2001-307982P P 20010725
US 2001-314356P P 20010822
US 2001-325020P P 20010925
US 2001-341746P P 20011212
US 2002-362158P P 20020305

AB The invention relates to 227 newly discovered nucleic acid mols. and their encoded proteins assocd. with prostate cancer including pre-malignant conditions. The higher than normal level of expression of any of these markers or combination of these markers correlates with the presence of prostate cancer in a patient. The markers are identified by transcription profiling using RNA derived from clin. samples which were chosen based on disease state, prognostic and diagnostic criteria; screening was performed with two custom arrays consisting of 6144 spots per membrane, including over 6000 subtracted library clones, more than 5000 IMAGE clones, and 200 control clones. Methods are provided for detecting the presence of cancer cancer in a sample, the absence of prostate cancer including pre-malignant conditions such as dysplasia in a sample, the stage of a prostate cancer, and with other characteristics of prostate cancer that are relevant to

L8 ANSWER 5 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2003:42302 CAPLUS
DOCUMENT NUMBER: 138:105633
TITLE: Tie-Pc and Ephrin-Fc fusion proteins for screening therapeutic capable of modulating growth, migration and proliferation of endothelial cells and treating cancers
INVENTOR(S): Alitalo, Kari; Kubo, Hajime
PATENT ASSIGNEE(S): Licentia Ltd., Finland
SOURCE: PCT Int. Appl., 200 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003004529	A2	20030116	WO 2002-IB2524	20020702

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-302960P P 20010702
AB The present invention provides materials and methods relating to the interaction between Tie receptors and Ephrin polypeptides. Methods based on the binding of Tie receptor and ephrin are useful for screening modulators of endothelial cell growth, migration and proliferation. These selected angiogenesis modulators, e.g. antibodies, are useful for treating cancers.

L8 ANSWER 6 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2003:42056 CAPLUS
DOCUMENT NUMBER: 138:67952
TITLE: Gene expression profiles useful for diagnosis of human bladder cancer and screening for modulators of bladder cancer
INVENTOR(S): Mack, David H.; Aziz, Natasha
PATENT ASSIGNEE(S): EOS Biotechnology, Inc., USA
SOURCE: PCT Int. Appl., 307 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 21
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003003906	A2	20030116	WO 2002-US21338	20020703
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
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WO 2003003906	A2	20030116	WO 2002-XA21338	20020703
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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2003003906	A2	20030116	WO 2002-XB21338	20020703
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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.:
US 2001-302814P P 20010703
US 2001-310099P P 20010803

L8 ANSWER 6 OF 67 CAPLUS COPYRIGHT 2003 ACS (Continued)
US 2001-343705P P 20011108
US 2001-350666P P 20011113
US 2002-372246P P 20020412
WO 2002-US21338 W 20020703

AB Described herein are genes whose expression are up-regulated or down-regulated in bladder cancer compared to normal adult tissues. The genes are identified using the Affymetrix/Eos Hu03 GeneChip microarrays contg. 59,680 probesets. Related methods and compns. that can be used for diagnosis and treatment of bladder cancer are disclosed. Also described herein are methods that can be used to identify modulators of bladder cancer. [This abstr. record is one of three records for this document necessitated by the large no. of index entries required to fully index the document and publication system constraints.].

L8 ANSWER 7 OF 67 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 1
ACCESSION NUMBER: 2002:285562 CAPLUS
DOCUMENT NUMBER: 137:61578
TITLE: Expressed gene sets as markers for specific tumors
INVENTOR(S): Ramaswamy, Sridhar; Golub, Todd B.; Tamayo, Pablo; Angelo, Michael
PATENT ASSIGNEE(S): Whitehead Institute for Biomedical Research, USA; Dana-Farber Cancer Institute, Inc.
SOURCE: PCT Int. Appl., 715 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002024956	A2	20020328	WO 2001-XB29287	20010919
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2002024956	A2	20020328	WO 2001-US29287	20010919
WO 2002024956	C1	20030306		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.:
US 2000-233534P P 20000919
US 2001-278749P P 20010326
WO 2001-US29287 W 20010919

L8 ANSWER 7 OF 67 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 1 (Continued)

AB Sets of genetic markers for specific tumor classes are described, as well as methods of identifying a biol. sample based on these markers. Total RNA was isolated from .apprx.300 human tumor and normal tissue specimens representing 30 individual classes of tumor or normal tissue, and cDNA produced using established mol. biol. protocols was hybridized to two high d. Affymetrix oligonucleotide microarrays (Hu6800FL and Hu35KsubA0). Raw expression data was combined into a master data set contg. the expression values for between 6800 and 16,000 genes expressed by each individual sample. A filter was applied to this data set which only allows those genes expressed at 3-fold above baseline and with an abs. difference in expression value of 100 to pass. By comparing the sets of genes which are expressed specifically in one class of tumor (e.g., pancreatic adenocarcinoma) vs. its accompanying normal tissue (e.g., normal pancreas), sets of genes were detd. which are specific to various tumors and their normal tissue counterparts. Also described are diagnostic, prognostic, and therapeutic screening uses for these markers, as well as oligonucleotide arrays comprising these markers. [This abstr. record is one of 4 records for this document necessitated by the large no. of index entries required to fully index the document and publication system constraints.].

L8 ANSWER 8 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:977957 CAPLUS
DOCUMENT NUMBER: 138:71917
TITLE: Antibodies specific to receptor protein tyrosine kinase for screening antibody phage library and for treating skeletal dysplasia, craniosynostosis, cell proliferative diseases or tumor
INVENTOR(S): Yayon, Avner; Rom, Eran
PATENT ASSIGNEE(S): Prochon Biotech Ltd., Israel
SOURCE: PCT Int. Appl., 122 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002102973	A2	20021227	WO 2002-IL495	20020620

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-299187P P 20010620
AB Mols. contg. the antigen-binding portion of antibodies that block constitutive and/or ligand-dependent activation of a receptor protein tyrosine kinase, such as fibroblast growth factor receptor 3 (FGFR3), are found through screening methods, where a sol. dimeric form of a receptor protein tyrosine kinase is used as target for screening a library of antibody fragments displayed on the surface of bacteriophage. The mols. of the present invention which block constitutive activation can be administered to treat or inhibit skeletal dysplasia, craniosynostosis disorders, cell proliferative diseases or disorders, or tumor progression assocd. with the constitutive activation of a receptor protein tyrosine kinase.

L8 ANSWER 9 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:977956 CAPLUS
DOCUMENT NUMBER: 138:71916
TITLE: Antibodies specific to receptor protein tyrosine kinase for screening antibody phage library and for treating skeletal dysplasia, craniosynostosis, cell proliferative diseases or tumor
INVENTOR(S): Yayon, Avner; Rom, Eran; Thomassen-Wolf, Elisabeth; Borges, Eric
PATENT ASSIGNEE(S): Prochon Biotech Ltd., Israel; Morphosys A.-G.
SOURCE: PCT Int. Appl., 122 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002102972	A2	20021227	WO 2002-IL494	20020620

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-299187P P 20010620
AB Mols. contg. the antigen-binding portion of antibodies that block constitutive and/or ligand-dependent activation of a receptor protein tyrosine kinase, such as fibroblast growth factor receptor 3 (FGFR3), are found through screening methods, where a sol. dimeric form of a receptor protein tyrosine kinase is used as target for screening a library of antibody fragments displayed on the surface of bacteriophage. The mols. of the present invention which block constitutive activation can be administered to treat or inhibit skeletal dysplasia, craniosynostosis disorders, cell proliferative diseases or disorders, or tumor progression assocd. with the constitutive activation of a receptor protein tyrosine kinase.

L8 ANSWER 10 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:977865 CAPLUS
DOCUMENT NUMBER: 138:71915
TITLE: Antibodies specific to receptor protein tyrosine kinase for screening antibody phage library and for treating skeletal dysplasia, craniosynostosis, cell proliferative diseases or tumor
INVENTOR(S): Thomassen-Wolf, Elisabeth; Borges, Eric; Yayon, Avner; Rom, Eran
PATENT ASSIGNEE(S): Morphosys A.-G., Germany; Prochon Biotech Ltd.
SOURCE: PCT Int. Appl., 103 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002102854	A2	20021227	WO 2002-IB3523	20020620

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-299187P P 20010620
AB Mols. contg. the antigen-binding portion of antibodies that block constitutive and/or ligand-dependent activation of a receptor protein tyrosine kinase, such as fibroblast growth factor receptor 3 (FGFR3), are found through screening methods, where a sol. dimeric form of a receptor protein tyrosine kinase is used as target for screening a library of antibody fragments displayed on the surface of bacteriophage. The mols. of the present invention which block constitutive activation can be administered to treat or inhibit skeletal dysplasia, craniosynostosis disorders, cell proliferative diseases or disorders, or tumor progression assocd. with the constitutive activation of a receptor protein tyrosine kinase.

L8 ANSWER 11 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2003:24612 CAPLUS
DOCUMENT NUMBER: 138:50950
TITLE: Gene expression profiles useful for diagnosis of human ovarian cancer and screening for modulators of ovarian cancer
INVENTOR(S): Mack, David H.; Gish, Kurt C.
PATENT ASSIGNEE(S): Eos Biotechnology Inc., USA
SOURCE: PCT Int. Appl., 332 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 21
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002102235	A2	20021227	WO 2002-XC19297	20020618

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

WO 2002102235	A2	20021227	WO 2002-US19297 20020618

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:
US 2001-299234P P 20010618
US 2001-315287P P 20010827
US 2001-317544P P 20010905
US 2001-350666P P 20011113
US 2002-372246P P 20020412
WO 2002-US19297 W 20020618

AB Described herein are genes whose expression are up-regulated or down-regulated in ovarian cancer compared to normal adult tissues. The genes are identified using the Affymetrix/Eos Hu01 or Hu03 GeneChip microarrays contg. 35,403 and 59,680 probesets, resp. Related methods and compns. that can be used for diagnosis and treatment of ovarian cancer are disclosed. Also described herein are methods that can be used to identify modulators of ovarian cancer. [This abstr. record is one of five records for this document necessitated by the large no. of index entries required to fully index the document and publication system constraints.].

L8 ANSWER 12 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:964539 CAPLUS
DOCUMENT NUMBER: 138:34222
TITLE: Differentially expressed human genes and their encoded proteins useful for identification, assessment, prevention, and therapy of cervical cancer
INVENTOR(S): Schlegel, Robert; Chen, Yan; Zhao, Xumei; Monahan, John E.; Kamatkar, Shubhangi; Gannavarapu, Manjula; Glatt, Karen; Hoersch, Sebastian
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 386 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002101075	A2	20021219	WO 2002-US18638	20020612

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:			
US 2001-298155P	P	20010613	
US 2001-298159P	P	20010613	
US 2001-335936P	P	20011114	

AB The invention relates to 119 newly discovered nucleic acid mols. and proteins assocd. with cervical cancer including pre-malignant conditions such as dysplasia in human patients. Cervical tumor-specific cDNA clones were identified by transcription profiling using mRNA from 12 cervical tumors, 5 CIN III, 5 CIN I, and 12 normal cervical tissues. The top up-regulated clones in tumors or DIN III cervical tissues, as detd. by proprietary statistical anal. methods, were selected, and full-length clones obtained by contiguous assembly of EST sequences. Compns., kits, and methods for detecting, characterizing, preventing, and treating human cervical cancers are provided.

L8 ANSWER 13 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:869047 CAPLUS
DOCUMENT NUMBER: 137:368581
TITLE: NOVX proteins, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated diseases such as cancer
INVENTOR(S): Alsobrook, John P., II; Anderson, David W.; Boldog, Ferenc L.; Burgess, Catherine E.; Casman, Stacie J.; Chapoval, Andrei; Edinger, Schlomit; Gerlach, Valerie; Gorman, Linda; Gunther, Erik; Guo, Xiaojia; Kekuda, Ramesh; Lepley, Denise M.; Li, Li; Liu, Xiaohong; Malyankar, Uriel M.; Miller, Charles E.; Millet, Isabelle; Padigaru, Muralidhara; Patturajan, Meera; Pena, Carol E. A.; Rieger, Daniel K.; Shenoy, Suresh G.; Shimkets, Richard A.; Spytek, Kimberly A.; Taupier, Raymond J., Jr.; Vernet, Corine A. M.; Voss, Edward Z.; Zerhusen, Bryan D.
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 340 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002090504	A2	20021114	WO 2002-US14342	20020502

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:			
US 2001-288395P	P	20010503	
US 2001-288900P	P	20010504	
US 2001-289087P	P	20010507	
US 2001-290753P	P	20010514	
US 2001-291189P	P	20010515	
US 2001-291243P	P	20010516	
US 2001-292001P	P	20010518	
US 2001-292374P	P	20010521	
US 2001-292587P	P	20010522	
US 2001-293107P	P	20010523	
US 2001-294110P	P	20010529	
US 2001-294434P	P	20010530	
US 2001-294827P	P	20010531	
US 2001-298988P	P	20010618	
US 2001-308901P	P	20010731	
US 2001-313388P	P	20010817	
US 2001-313851P	P	20010821	
US 2001-313937P	P	20010821	

L8 ANSWER 13 OF 67 CAPLUS COPYRIGHT 2003 ACS (Continued)

US 2001-322701P	A1	20010917	
US 2001-322701P	P	20010917	
US 2001-322802P	P	20010917	
US 2001-324757P	P	20010925	
US 2001-325314P	P	20010927	
US 2001-325682P	P	20010927	
US 2001-332129P	P	20011121	
US 2001-336882P	P	20011203	
US 2001-340305P	P	20011214	
US 2002-138588	P	20020501	

AB Disclosed herein are nucleic acid sequences that encode antigenic NOVX polypeptides, i.e. NOV1a, NOV2a, NOV2b, NOV2c, NOV3, NOV4a, NOV4b, NOV5, NOV6a, NOV6b, NOV7, NOV8a, NOV8b, NOV8c, NOV8d, NOV9, NOV10a, NOV10b, NOV11, NOV12a, NOV12b, NOV13a, NOV13b, NOV13c, NOV13d, NOV14, NOV15a, NOV15b, NOV16, NOV17, NOV18a, NOV18b, NOV19a, NOV19b, NOV20a, NOV20b, NOV21, NOV22a, NOV22b, NOV23a, NOV23b, NOV24, NOV25, NOV26a, NOV26b, and NOV27 proteins and genes. Also disclosed are antibodies, which immunospecifically-bind to the NOVX polypeptide, as well as derivs., variants, mutants, or fragments of the aforementioned polypeptide, polynucleotide, or antibody. The invention further discloses therapeutic, diagnostic and research methods for diagnosis, treatment, and prevention of disorders involving any one of these novel human nucleic acids, polypeptides, or antibodies, or fragments thereof. The disorders include metabolic diseases, diabetes, obesity, infection, anorexia, cancer-assocd. cachexia, cancer, neurodegenerative diseases, Alzheimer's disease, Parkinson's disease, immune diseases, and hematopoietic diseases.

L8 ANSWER 14 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:615889 CAPLUS
DOCUMENT NUMBER: 137:180730
TITLE: Human cDNA/DNA molecules and proteins encoded by them with enhanced expression in apoptosis-resistant cell clones, and use thereof in diagnosis, therapeutics and drug screening
INVENTOR(S): Ullrich, Axel; Abraham, Reimar
PATENT ASSIGNEE(S): Max-Planck-Gesellschaft zur Foerderung der Wissenschaften e.V., Germany
SOURCE: PCT Int. Appl., 56 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002063037	A2	20020815	WO 2002-EP1073	20020201

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:			
US 2001-265631P	P	20010202	

AB The present invention relates to a method for identifying nucleic acid mols. functionally assocd. with a desired phenotype, such as cancer cell properties, including anti-apoptosis. The method, which allows for generation of expression profiles of genes assocd. with said desired phenotype, involves a mutagenesis and/or genome rearrangement step, followed by selection of cell clones displaying the desired phenotype. The invention also relates that the method involves the use of the following techniques: fluorescence-activated cell sorting (FACS); nucleic acid microarray (cDNA, genomic or oligonucleotide); protein array; two-dimensional gel electrophoresis; and/or mass spectrometry. The invention further relates that the disclosed method was used to identify genes, which are differentially expressed in apoptosis-sensitive and apoptosis-resistant cells. Specifically, the invention relates that apoptosis was induced in human cervix carcinoma cell line HeLa S3 by Fas activation. After the selection procedure, only a low no. of living cells were present, which had a higher resistance against apoptosis than the parental cell line. MRNA was isolated from these surviving clones, and from the parental cell line, and transcribed into cDNA. CDNA microarray technol. was used to identify about 150-200 genes (cDNA/DNA mols.) that exhibited enhanced expression in apoptosis-resistant clones. The GenBank accession nos. of some of these cDNA/DNA mols. are provided, along with the products encoded by said mols. Still further, the invention relates that most of the apoptosis-assocd. genes encode protein phosphatases, and kinases. Finally, the invention relates that said nucleic acid mols., and proteins encoded by mols., can be used as targets in diagnosis, therapeutics and drug screening, particularly for disorders assocd. with dysfunction of apoptotic processes, such as tumors.

L8 ANSWER 14 OF 67 CAPLUS COPYRIGHT 2003 ACS (Continued)

L8 ANSWER 15 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:429201 CAPLUS
DOCUMENT NUMBER: 137:4997
TITLE: Method for diagnosing allergic diseases using DNA and protein microarray technology
INVENTOR(S): Schmidt-Weber, Carsten; Blaser, Kurt; Wohlfahrt, Jan
PATENT ASSIGNEE(S): Genescan Europe Ag, Germany
SOURCE: PCT Int. Appl., 61 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002044732	A2	20020606	WO 2001-EP13937	20011129
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1221618	A1	20020710	EP 2000-126117	20001129
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
AU 2002021906	A5	20020611	AU 2002-21906	20011129

PRIORITY APPLN. INFO.: EP 2000-126117 A 20001129
WO 2001-EP13937 W 20011129

AB MRNA of activated lymphocytes such as CD4+ T cells allows differential diagnosis of allergic diseases. The CD4+ T cells are isolated and stimulated under defined conditions in vitro. Subsequently, mRNA is subjected to multigene anal. such as DNA arrays. Expression profiling images, such as gene expression profiles, can be created, which allow on the basis of the activated T cell mRNA the prediction of certain phenotypes such as asthma or atopic dermatitis.

L8 ANSWER 16 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:391768 CAPLUS
DOCUMENT NUMBER: 136:382014
TITLE: Artery and vein smooth muscle-specific Ephrin family of ligands as molecular markers and uses
INVENTOR(S): Anderson, David J.; Garcia-Cardena, Guillermo; Gimbrone, Michael A., Jr.; Wang, Hai U.
PATENT ASSIGNEE(S): California Institute of Technology, USA; The Brigham and Women's Hospital, Inc.
SOURCE: PCT Int. Appl., 82 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002040540	A2	20020523	WO 2001-US42961	20011120
WO 2002040540	A3	20030116		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002032405	A5	20020527	AU 2002-32405	20011120
US 2002136726	A1	20020926	US 2001-988496	20011120

PRIORITY APPLN. INFO.: US 2000-252009P P 20001120
WO 2001-US42961 W 20011120

AB The present invention relates to methods of distinguishing and sepg. arterial cells from venous cells, and more specifically, distinguishing and sepg. arterial smooth muscle cells from venous smooth muscle cells based on their resp. mol. markers; methods of selectively targeting or delivering agents, drugs, nucleic acids and/or gene products to arteries (and in particular to arterial smooth muscle cells) or veins; methods of altering (enhancing or inhibiting, where inhibiting includes partially or completely inhibiting) the function of artery-specific or vein-specific mol. markers or interaction between them (and, thus, enhancing or inhibiting the effect such functions or interactions have on arterial smooth muscle cells or venous smooth muscle cells); and methods of screening for drugs which act selectively on arterial cells (and more specifically, on arterial smooth muscle cells) or venous cells (and more specifically, on venous smooth muscle cells). In one embodiment the mol. marker is a member of a smooth muscle cell surface ligand-receptor pair which is differentially expressed on arterial and venous smooth muscle cells. For example, as described in detail herein, a member of the Ephrin family of ligands is a mol. marker for arterial smooth muscle cells and can be used to distinguish or isolate arterial smooth muscle cells. Expression of EphrinB2 in arterial cells (e.g., arterial endothelial cells, arterial smooth muscle cells) can be used to advantage in methods for targeting agents and/or encoded polypeptides to arterial smooth muscle cells, altering angiogenesis, assessing the effect of agents on arterial smooth muscle cells, identifying arterial smooth muscle cells, isolating arterial smooth muscle cells and prodn. of artificial vessels, for

L8 ANSWER 16 OF 67 CAPLUS COPYRIGHT 2003 ACS (Continued)

example. Protein. The transmembrane ligand ephrinB2 and its receptor tyrosine kinase EphB4 are mol. markers of embryonic arterial and venous endothelial cells, resp., and are essential for angiogenesis. Here the authors show that expression of ephrinB2 persists in adult arteries where it extends into some of the smallest diam. microvessels, challenging the classical view that capillaries have neither arterial nor venous identity.

EphrinB2 also identifies arterial microvessels in several settings of adult neovascularization, including tumor angiogenesis, contravening the dogma that tumor vessels arise exclusively from postcapillary venules. Unexpectedly, expression of ephrinB2 also defines a stable genetic difference between arterial and venous vascular smooth muscle cells. These observations argue for revisions of classical concepts of capillary identity and the topog. of neovascularization. They also imply that ephrinB2 may be functionally important in neovascularization and in arterial smooth muscle, as well as in embryonic angiogenesis.

L8 ANSWER 17 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:293699 CAPLUS
DOCUMENT NUMBER: 136:320393
TITLE: Sequence homologs of thymosin .beta.10, ephrin A8 receptors and fibromodulin and cDNA encoding them and theor therapeutic uses
INVENTOR(S): Prayaga, Sudhirdas K.; Taupier, Raymond J.; Bandaru, Raj
PATENT ASSIGNEE(S): Curagen Corporation, USA
SOURCE: PCT Int. Appl., 180 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002030979	A2	20020418	WO 2001-US31498	20011010
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002011543	A5	20020422	AU 2002-11543	20011010

PRIORITY APPLN. INFO.:

US 1999-159805P	P	19991015
US 1999-159992P	P	19991018
US 1999-86423P	P	19991018
US 1999-160952P	P	19991022
US 2000-689486	A2	20001012
US 2000-687276	A2	20001013
US 2001-973424	A	20011009
WO 2001-US31498	W	20011010

AB Disclosed herein are novel human nucleic acid sequences that have homol. to thymosin, ephrin A receptors, proteoglycans and fibromodulin. Also disclosed are polypeptides encoded by these nucleic acid sequences, and antibodies which immunospecifically-bind to the polypeptide, as well as derivs., variants, mutants, or fragments of the aforementioned polypeptide, polynucleotide, or antibody. The invention further discloses therapeutic, diagnostic and research methods for diagnosis, treatment, and prevention of disorders involving these novel nucleic acids and proteins.

L8 ANSWER 18 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:256329 CAPLUS
DOCUMENT NUMBER: 136:273231
TITLE: Extracellular domains of Eph B4 receptor and ephrin B2 ligand, their sequences, recombinant production, and therapeutic uses
INVENTOR(S): Martiny-Baron, Georg; Wood, Jeanette Marjorie; Liau, Gene
PATENT ASSIGNEE(S): Novartis A.-G., Switz.; Novartis-Erfindungen Verwaltungsgesellschaft m.b.H.
SOURCE: PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002026827	A1	20020404	WO 2001-EP11252	20010928
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002012292	A5	20020408	AU 2002-12292	20010928

PRIORITY APPLN. INFO.:

CH 2000-1910	A	20000929
WO 2001-EP11252	W	20010928

AB The invention provides polypeptides comprising the sol. extracellular domain of Eph B4 receptor or ephrin B2 ligand from human and mouse. The invention relates that Eph receptors are members of a receptor tyrosine kinase family. The invention also provides cDNA mols. encoding said polypeptides. The invention further provides genetic vectors, such as viral vectors, contg. said cDNA mols., and host cells transformed with said vectors. Still further, the invention provides pharmaceutical compns. comprising said polypeptides, cDNA mols. and/or genetic vectors, for treating a mammalian disease, such as cancer or ocular neovascularization. Finally, the invention provides the cDNA sequences, as well as amino acid sequences, of the extracellular domains of Eph B4 receptor and ephrin B2 ligand from mouse and human. The invention was based on the findings that Eph B receptors and ephrin B ligands participate in **tumor** angiogenesis in adult mammals, and that said polypeptides were suitable for treatment of ocular neovascularization. In the examples section, the invention specifically demonstrated inhibition of: (1) **tumor** angiogenesis and **tumor** growth by expression of the sol. extracellular domains of mouse Eph B4 or ephrin B2 in **tumor** cells; and (2) both VEGF and FGF-2-directed angiogenesis by expression of human Eph B4, using gene therapy vector AV3Cshs-EphB4.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 18 OF 67 CAPLUS COPYRIGHT 2003 ACS (Continued)

L8 ANSWER 19 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:493975 CAPLUS
DOCUMENT NUMBER: 137:52335
TITLE: Manipulation of arterial-venous identity
INVENTOR(S): Li, Dean Y.
PATENT ASSIGNEE(S): The University of Utah Research Foundation, USA
SOURCE: PCT Int. Appl., 39 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002011785	A2	20020214	WO 2001-US24405	20010803
WO 2002011785	A3	20020530		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2002081284	A1	20020627	US 2001-921771	20010803

PRIORITY APPLN. INFO.:

US 2000-222759P	P	20000803

AB Methods and compns. for manipulating the arterial-venous identity of endothelial cells are provided. The methods comprise introducing an arterial mol. program into endothelial cells of a vein section such that the endothelial cells can remodel to form arterial endothelial cells. The arterial mol. program can comprise one or more polynucleotides encoding various genes that are assocd. with arterial development and/or differentiation from veins. Expression vectors comprising the genes can be used to introduce the mol. program into the cells. A method of treating a patient having an obstructed blood vessel is also provided.

L8 ANSWER 20 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:553118 CAPLUS
DOCUMENT NUMBER: 137:119642
TITLE: RT-PCR based methods for determining cancer treatment efficacy using expression profiles of marker genes
INVENTOR(S): Van der Kuyl, Antoinette Cornelia; Cornelissen, Marion
PATENT ASSIGNEE(S): Amsterdam Support Diagnostics B.V., Neth.
SOURCE: Eur. Pat. Appl., 38 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1225233	A2	20020724	EP 2002-75264	20020123
EP 1225233	A3	20030108		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
EP 1298221	A1	20030402	EP 2001-203703	20010928
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

PRIORITY APPLN. INFO.: EP 2001-200228 A 20010123
EP 2001-203703 A 20010928
US 2001-325722P P 20010928

AB The invention provides a method for detg. whether a treatment is effective in changing the status of a certain set of target cells, such as a tumor, in a patient. This method implies obtaining a sample from a patient after initiation of a treatment, and detg. whether said sample comprises an expression product of at least one marker gene. Preferably, said sample is a blood sample. In one aspect, said expression product is expressed by a peripheral blood mononuclear cell. Said marker gene may be a gene involved in the generation, maintenance and/or breakdown of blood vessels (angiogenesis). A method of the invention is very suitable to det. within a few days if a certain treatment against Kaposi's Sarcoma is successful. Moreover, this method is suitable for detg. the presence of angiogenesis and/or tumor cells in a patient.

L8 ANSWER 21 OF 67 USPATFULL
ACCESSION NUMBER: 2002:307817 USPATFULL
TITLE: Methods and reagents for isolating biologically active peptides
INVENTOR(S): Gyuris, Jeno, Winchester, MA, UNITED STATES
Morris, Aaron J., Boston, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002172940	A1	20021121
APPLICATION INFO.:	US 2002-80854	A1	20020222 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1998-174943, filed on 19 Oct 1998, GRANTED, Pat. No. US 6420110
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ROPES & GRAY, ONE INTERNATIONAL PLACE, BOSTON, MA, 02110-2624
NUMBER OF CLAIMS: 79
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 14 Drawing Page(s)
LINE COUNT: 3210
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB One aspect of the present invention is the synthesis of a binary method that combines variegated peptide display libraries, e.g., in a "display mode", with soluble secreted peptide libraries, e.g., in a "secretion mode", to yield a method for the efficient isolation of peptides having a desired biological activity.

L8 ANSWER 22 OF 67 USPATFULL
ACCESSION NUMBER: 2002:250788 USPATFULL
TITLE: Artery smooth muscle- and vein smooth muscle-specific proteins and uses therefor
INVENTOR(S): Anderson, David J., Atladena, CA, UNITED STATES
Garcia-Cardena, Guillermo, Boston, MA, UNITED STATES
Gimbrone, Michael A., JR., Jamaica Plain, MA, UNITED STATES
Wang, Hai U., Eldorado Hills, CA, UNITED STATES
PATENT ASSIGNEE(S): California Institute of Technology, Pasadena, CA (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002136726	A1	20020926
APPLICATION INFO.:	US 2001-988496	A1	20011120 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-252009P	20001120 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HAMILTON, BROOK, SMITH & REYNOLDS, P.C., 530 VIRGINIA ROAD, P.O. BOX 9133, CONCORD, MA, 01742-9133
NUMBER OF CLAIMS: 72
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Page(s)
LINE COUNT: 2825
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Arterial and venous smooth muscle cells are molecularly distinct from the earliest stages of angiogenesis through to adulthood. This distinction is revealed by expression on arterial cells (e.g., arterial endothelial cells, arterial smooth muscle cells) of a transmembrane ligand, called EphrinB2 whose receptor EphB4 is expressed on venous cells. Targeted disruption of the EphrinB2 gene prevents the remodeling of veins from a capillary plexus into properly branched structures. Moreover, it also disrupts the remodeling of arteries, suggesting that reciprocal interactions between pre-specified arterial and venous cells are necessary for angiogenesis. Expression of EphrinB2 in arterial cells (e.g., arterial endothelial cells, arterial smooth muscle cells) can be used to advantage in methods for targeting agents and/or encoded polypeptides to arterial smooth muscle cells, altering angiogenesis, assessing the effect of agents on arterial smooth muscle cells, identifying arterial smooth muscle cells, isolating arterial smooth muscle cells and production of artificial vessels, for example.

L8 ANSWER 23 OF 67 USPATFULL
ACCESSION NUMBER: 2002:156689 USPATFULL
TITLE: Manipulation of arterial-venous identity
INVENTOR(S): Li, Dean Y., Salt Lake City, UT, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002081284	A1	20020627
APPLICATION INFO.:	US 2001-921771	A1	20010803 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-222759P	20000803 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: J. Matthew Buchanan, BRINKS HOFER GILSON & LIONE, P.O. Box 10395, Chicago, IL, 60610
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
LINE COUNT: 943
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods and compositions for manipulating the arterial-venous identity of endothelial cells are provided. The methods comprise introducing an arterial molecular program into endothelial cells of a vein section such that the endothelial cells can remodel to form arterial endothelial cells. The arterial molecular program can comprise one or more polynucleotides encoding various genes that are associated with arterial development and/or differentiation from veins. Expression vectors comprising the genes can be used to introduce the molecular program into the cells. A method of treating a patient having an obstructed blood vessel is also provided.

L8 ANSWER 24 OF 67 USPATFULL
ACCESSION NUMBER: 2002:133509 USPATFULL
TITLE: METHODS OF EVALUATING SPECIFIC CELLULAR FUNCTIONS OF RECEPTOR PROTEIN TYROSINE KINASES IN A LIGAND INDEPENDENT MANNER
INVENTOR(S): CLARY, DOUGLAS, SAN FRANCISCO, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002068361	A1	20020606
APPLICATION INFO.:	US 1998-57150	A1	19980407 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1997-43207P	19970408 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FOLEY & LARDNER, 3000K STREET NW, SUITE 500, WASHINGTON, DC, 20007
NUMBER OF CLAIMS: 26
EXEMPLARY CLAIM: 1
LINE COUNT: 1254
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to methods of evaluating the specific function of a receptor protein tyrosine kinase in cells. The methods activate the receptor in a ligand independent fashion. In addition, the invention includes methods of identifying compounds that modulate receptor protein tyrosine kinase function.

L8 ANSWER 25 OF 67 USPATFULL
ACCESSION NUMBER: 2002:43170 USPATFULL
TITLE: Methods and reagents for isolating biologically active antibodies
INVENTOR(S): Gyuris, Jeno, Winchester, MA, UNITED STATES
Ewert, Sebastian-Meier, Wolfratshausen, GERMANY, FEDERAL REPUBLIC OF
Nagy, Zolton, Wolfratshausen, GERMANY, FEDERAL REPUBLIC OF
Morris, Aaron, Brighton, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002025536	A1	20020228
APPLICATION INFO.:	US 2001-891557	A1	20010626 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-214200P	20000626 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ROPES & GRAY, ONE INTERNATIONAL PLACE, BOSTON, MA, 02110-2624
NUMBER OF CLAIMS: 83
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 3051
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB One aspect of the present invention is the synthesis of a binary method that combines variegated antibody display libraries, e.g., in a "display mode", with soluble secreted antibody libraries, e.g., in a "secretion mode", to yield a method for the efficient isolation of antibodies having a desired biological activity.

L8 ANSWER 26 OF 67 USPATFULL
ACCESSION NUMBER: 2002:17445 USPATFULL
TITLE: Cytokines that bind the cell surface receptor hek
INVENTOR(S): Beckmann, M. Patricia, Poulsbo, WA, UNITED STATES
Cerretti, Douglas P., Seattle, WA, UNITED STATES
PATENT ASSIGNEE(S): Immunex Corporation (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002010325	A1	20020124
APPLICATION INFO.:	US 2001-904954	A1	20010712 (9)
RELATED APPLN. INFO.:	Division of Ser. No. US 1999-358734, filed on 21 Jul 1999, GRANTED, Pat. No. US 6274117 Division of Ser. No. US 1998-57121, filed on 8 Apr 1998, GRANTED, Pat. No. US 5969110 Division of Ser. No. US 1995-453943, filed on 30 May 1995, GRANTED, Pat. No. US 5738844 Division of Ser. No. US 1994-240124, filed on 9 May 1994, GRANTED, Pat. No. US 5516658 Continuation-in-part of Ser. No. US 1993-161132, filed on 3 Dec 1993, ABANDONED Continuation-in-part of Ser. No. US 1993-114426, filed on 30 Aug 1993, ABANDONED Continuation-in-part of Ser. No. US 1993-109745, filed on 20 Aug 1993, ABANDONED		

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: IMMUNEX CORPORATION, LAW DEPARTMENT, 51 UNIVERSITY STREET, SEATTLE, WA, 98101
NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 1
LINE COUNT: 1713
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Hek ligand (hek-L) polypeptides as well as DNA sequences, vectors and transformed host cells useful in providing hek-L polypeptides. The hek-L polypeptides bind to a cell surface receptor (hek) that is a member of the receptor tyrosine kinase family. Hek is expressed on cells that include certain tumor cell lines. The hek-L polypeptides also bind a distinct receptor tyrosine kinase known elk.

L8 ANSWER 27 OF 67 USPATFULL
ACCESSION NUMBER: 2002:174944 USPATFULL
TITLE: Methods and reagents for isolating biologically active peptides
INVENTOR(S): Gyuris, Jeno, Winchester, MA, United States
Morris, Aaron J., Boston, MA, United States
PATENT ASSIGNEE(S): GPC Biotech, Inc., Waltham, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6420110	B1	20020716
APPLICATION INFO.:	US 1998-174943		19981019 (9)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Ponnaluri, Padmashri
LEGAL REPRESENTATIVE: Ropes & Gray, Vincent, Matthew P., Halstead, David P.
NUMBER OF CLAIMS: 42
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 17 Drawing Figure(s); 14 Drawing Page(s)
LINE COUNT: 3145
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB One aspect of the present invention is the synthesis of a binary method that combines variegated peptide display libraries, e.g., in a "display mode", with soluble secreted peptide libraries, e.g., in a "secretion mode", to yield a method for the efficient isolation of peptides having a desired biological activity.

L8 ANSWER 28 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:629172 CAPLUS
DOCUMENT NUMBER: 137:382746
TITLE: Regulation of **vasculogenesis** and angiogenesis by EphB/ephrin-B2 signaling between endothelial cells and surrounding mesenchymal cells
AUTHOR(S): Oike, Yuichi; Ito, Yasuhiro; Hamada, Koichi; Zhang, Xiu-Qin; Miyata, Keishi; Arai, Fumio; Inada, Tomohisa; Araki, Kimi; Nakagata, Naomi; Takeya, Motohiro; Kisanuki, Yaz Y.; Yanagisawa, Masashi; Gale, Nicholas W.; Suda, Toshio
CORPORATE SOURCE: Department of Cell Differentiation, the Department of Developmental Genetics, the Institute of Molecular Embryology and Genetics, Kumamoto University, Kumamoto, Japan
SOURCE: Blood (2002), 100(4), 1326-1333
CODEN: BLOOAW; ISSN: 0006-4971
PUBLISHER: American Society of Hematology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Although the cellular and mol. mechanisms governing angiogenesis are only beginning to be understood, signaling through endothelial-restricted receptors, particularly receptor tyrosine kinases, has been shown to play a pivotal role in these events. Recent reports show that EphB receptor tyrosine kinases and their transmembrane-type ephrin-B2 ligands play essential roles in the embryonic **vasculature**. These studies suggest that cell-to-cell repellent effects due to bidirectional EphB/ephrin-B2 signaling may be crucial for **vascular** development, similar to the mechanism described for neuronal development. To test this hypothesis, we disrupted the precise expression pattern of EphB/ephrin-B2 in vivo by generating transgenic (CAGp-ephrin-B2 Tg) mice that express ephrin-B2 under the control of a ubiquitous and constitutive promoter, CMV enhancer-.beta.-actin promoter-.beta.-globin splicing acceptor (CAG). These mice displayed an abnormal segmental arrangement of intersomitic vessels, while such anomalies were not obsd. in Tie-2p-ephrin-B2 Tg mice in which ephrin-B2 was overexpressed in only **vascular** endothelial cells (ECs). This finding suggests that non-ECs expressing ephrin-B2 alter the migration of ECs expressing EphB receptors into the intersomitic region where ephrin-B2 expression is normally absent. CAGp-ephrin-B2 Tg mice show sudden death at neonatal stages from aortic dissecting aneurysms due to defective recruitment of **vascular** smooth muscle cells to the ascending aorta. EphB/ephrin-B2 signaling between endothelial cells and surrounding mesenchymal cells plays an essential role in **vasculogenesis**, angiogenesis, and vessel maturation.
REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 29 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:815692 CAPLUS
DOCUMENT NUMBER: 137:309445
TITLE: EphB6 crosslinking results in costimulation of T cells
AUTHOR(S): Luo, Hongyu; Yu, Guang; Wu, Yulian; Wu, Jiangping
CORPORATE SOURCE: Laboratory of Transplantation Immunology, Centre Hospitalier de l'Universite de Montreal, University of Montreal, Montreal, QC, H2L 4M1, Can.
SOURCE: Journal of Clinical Investigation (2002), 110(8), 1141-1150
CODEN: JCINAO; ISSN: 0021-9738
PUBLISHER: American Society for Clinical Investigation
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Erythropoietin-producing hepatocyte (Eph) kinases represent the largest receptor tyrosine kinase family. Some of them are expressed in the T cell compartment, but their function in T cells is unknown. In peripheral blood, EphB6 was predominantly expressed on T cells, and was upregulated after culture. EphB6 crosslinking by anti-EphB6 mAb or ephrinB2 in the presence of suboptimal T cell receptor (TCR) stimulation led to drastic T cell proliferation, suggesting that EphB6 can costimulate T cells. The proliferation was accompanied by enhanced prodn. of several lymphokines, such as IFN-.gamma., IL-6, IL-10, TGF-.beta., TNF-.alpha., and GM-CSF, but not IL-2 and IL-4. Sorted EphB6+ T cells had significantly stronger response to anti-CD3 and anti-CD28 stimulation than EphB6- T cells had. Taken together, these data suggest an important role of EphB6 in normal T cell activation. Within two minutes of anti-CD3 and anti-CD28 stimulation, EphB6 aggregated and colocalized with TCR, and this provides a morphol. basis for EphB6 to enhance TCR signaling. The capping was followed by p38 MAPK activation, showing that EphB6 is capable of signaling, in spite of its lack of intrinsic kinase activity. This study demonstrates that interaction between EphB6 and its ligands facilitates T cell responses to antigen.
REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 30 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:534292 CAPLUS
DOCUMENT NUMBER: 137:242444
TITLE: EphB ligand, ephrinB2, suppresses the VEGF- and angiopoietin-1-induced Ras/mitogen-activated protein kinase pathway in venous endothelial cells
AUTHOR(S): Kim, Injune; Ryu, Young Shin; Kwak, Hee Jin; Ahn, So Young; Oh, Jong-Lark; Yancopoulos, George D.; Gale, Nicholas W.; Koh, Gou Young
CORPORATE SOURCE: National Creative Research Initiatives Center for Endothelial Cells and Department of Life Science, Division of Molecular and Life Sciences, Pohang University of Science and Technology, Pohang, 790-784, S. Korea
SOURCE: FASEB Journal (2002), 16(9), 1126-1128, 10.1096/fj.01-0805fje
CODEN: FAJOEC; ISSN: 0892-6638
PUBLISHER: Federation of American Societies for Experimental Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Interaction between ephrinB2 and EphB4 in endothelial cells at the arterial-venous capillary interface is crit. for proper embryonic capillary morphogenesis. However, the intracellular downstream signaling of ephrinB2-EphB in **vascular** endothelial cells is unknown. This study examd. the effect of ephrinB2-induced activation of EphB kinases on **vascular** endothelial growth factor (VEGF)- and angiopoietin-1 (Ang1)-induced Ras/mitogen-activated protein kinase (MAPK) signaling cascades in human umbilical vein endothelial cells (HUVECs). Reverse transcriptase-polymer chain reaction results showed that HUVECs expressed three kinds of EphB kinases known to bind to ephrinB2: EphB2, EphB3, and EphB4. EphrinB2 not only increased the phosphorylation of EphB2 and EphB4 in a time-dependent manner but also increased recruitment of p120-Ras-GTPase-activating protein (p120-RasGAP) to EphB2 and EphB4. Accordingly, ephrinB2 inhibited VEGF- and Ang1-induced Ras-MAPK activities, whereas ephrinB2 did not alter VEGF-induced Flk phosphorylation or Ang1-induced Tie2 phosphorylation. Furthermore, ephrinB2 suppressed VEGF- and Ang1-induced proliferation and/or migration, which are mediated mainly through Ras/MAPK signaling cascades. From these results, the authors propose that ephrinB2-EphB, signaling through Ras/MAPK cascade, may be crit. for proper morphogenesis of capillary endothelium through the arrest of endothelial cell proliferation and migration at the arterial-venous interface.
REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 31 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:435872 CAPLUS
DOCUMENT NUMBER: 137:211279
TITLE: A role of EphB4 receptor and its ligand, ephrin-B2, in erythropoiesis
AUTHOR(S): Suenobu, Souichi; Takakura, Nobuyuki; Inada, Tomohisa; Yamada, Yoshihiro; Yuasa, Hiromi; Zhang, Xiu-Qin; Sakano, Seiji; Oike, Yuichi; Suda, Toshio
CORPORATE SOURCE: Department of Cell Differentiation, Institute of Molecular Embryology and Genetics (IMEG), Kumamoto University, Tokyo, Japan
SOURCE: Biochemical and Biophysical Research Communications (2002), 293(3), 1124-1131
CODEN: BBRCA9; ISSN: 0006-291X
PUBLISHER: Elsevier Science
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Erythropoiesis is regulated not only by erythropoietin but also by microenvironments which are composed of transmembrane mols. We have previously shown that a receptor tyrosine kinase EphB4 is predominantly expressed on human erythroid progenitors in bone marrow. EphB4 is expressed in approx. 45% of hematopoietic progenitor cells, which are CD34-pos. and c-Kit-pos. cells in human umbilical cord blood (hUCB). The transmembrane ligand for EphB4, ephrin-B2, is expressed on bone marrow stromal cells and arterial endothelial cells. When such EphB4-pos. hematopoietic progenitor cells were co-cultured with stromal cells which express ephrin-B2, they were immediately detached from stromal cells and differentiated to mature erythroid cells. At that time, expression of EphB4 was immediately down-regulated. In contrast, on ephrin-B2 non-expressing stromal cells, they remained EphB4-pos. cells and the generated no. of mature erythroid cells was less than that on ephrin-B2 expressing stromal cells. Addnl., ephrin-B2 expression on endothelial cells was up-regulated under hypoxic condition. Taken together, we propose that one of the mol. cues that regulate erythropoiesis is ephrin-B2 on stromal cells.
REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 32 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:683384 CAPLUS
DOCUMENT NUMBER: 137:383059
TITLE: Loss of EphB4 receptor tyrosine kinase protein expression during carcinogenesis of the human breast
AUTHOR(S): Berclaz, Gilles; Fluetsch, Bettina; Altermatt, Hans Joerg; Rohrbach, Valeria; Djonov, Valentin; Ziemiecki, Andrew; Dreher, Ekkehard; Andres, Anne-Catherine
CORPORATE SOURCE: Department of Obstetrics and Gynecology, University Hospital, Inselspital Bern, Bern, Switz.
SOURCE: Oncology Reports (2002), 9(5), 985-989
CODEN: OCRPEW; ISSN: 1021-335X
PUBLISHER: Oncology Reports
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Members of the Eph family of receptor tyrosine kinase have been implicated in cell-cell communication and tissue integrity during embryogenesis. We have previously demonstrated cell type specific and hormone dependent EphB4 expression in the mouse mammary parenchyma suggesting involvement in the homeostasis of this organ. Since disruption of tissue organization is crucial for metastatic dissemination, we have investigated the expression of EphB4 during carcinogenesis of the human breast. Immunohistochem. anal. of 24 normal human breast samples and 124 consecutive breast carcinomas was correlated with tumor characteristics (stage, histol., grade, lymph node involvement) and the expression of ER, PR, Ki-67, p53 and HER2. In normal breast tissue, the EphB4 protein was expressed exclusively in parenchymal cells. Strikingly, a drastic redn. in the no. of EphB4 protein expressing cells was obsd. in almost all invasive carcinomas analyzed, irresp. of the tumor type ($p<0.0001$). Furthermore, we found a highly significant correlation between EphB4 positivity and low histol. grading of the tumor cells ($p=0.002$) suggesting that in breast cancer, EphB4 expression is not compatible with tumor progression. This raises the possibility that EphB4 could represent a potent tool for therapeutic intervention.
REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS FORMAT RECORD. ALL CITATIONS AVAILABLE IN THE RE

L8 ANSWER 33 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:209447 CAPLUS
DOCUMENT NUMBER: 137:137868
TITLE: Origin, differentiation, and maturation of human pulmonary veins
AUTHOR(S): Hall, Susan M.; Hislop, Alison A.; Haworth, Sheila G.
CORPORATE SOURCE: Unit of Vascular Biology and Pharmacology, Institute of Child Health, University College, London, UK
SOURCE: American Journal of Respiratory Cell and Molecular Biology (2002), 26(3), 333-340
CODEN: AJRBEL; ISSN: 1044-1549
PUBLISHER: American Thoracic Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Recent studies on human embryonic and fetal lungs show that the pulmonary arteries form by **vasculogenesis**. Little is known of the early development of the pulmonary veins. Using immunohistochem. techniques and serial reconstruction, the authors studied 18 fetal and neonatal lungs. Sections were stained with antibodies specific for endothelium (CD31, von Willebrand factor) and smooth muscle (.alpha. and .gamma. smooth muscle actin, smooth muscle myosin, calponin, caldesmon, and desmin) and antibodies specific for the matrix glycoprotein tenascin, the receptor protein tyrosine kinase EphB4, and its ligand ephrinB2. Kiel University-raised antibody no. 67 (Ki67) expression allowed qual. assessment of cell replication. By 34 d gestation, there was continuity between the aortic sac, pulmonary arteries, capillaries, pulmonary veins, and atrium. The pulmonary veins formed by **vasculogenesis** in the mesenchyme surrounding the terminal buds during the pseudoglandular period and probably by angiogenesis in the canalicular and alveolar stages. EphB4 and ephrinB2 did not distinguish between presumptive venous and arterial endothelium as they do in mouse. All venous smooth muscle cells derived directly from the mesenchyme, gradually acquiring smooth muscle specific proteins from 56 d gestation. Thus, both pulmonary arteries and veins arise by **vasculogenesis**, but the origins of their smooth muscle cells and their cytoskeletal protein content are different.
REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS FORMAT RECORD. ALL CITATIONS AVAILABLE IN THE RE

L8 ANSWER 34 OF 67 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 2
ACCESSION NUMBER: 2002:88305 CAPLUS
DOCUMENT NUMBER: 136:260508
TITLE: Altered mammary epithelial development, pattern formation and involution in transgenic mice expressing the EphB4 receptor tyrosine kinase
AUTHOR(S): Munarini, Nadia; Jager, Richard; Abderhalden, Susanne; Zuercher, Gisela; Rohrbach, Valeria; Loercher, Saemi; Pfanner-Meyer, Brigitte; Andres, Anne-Catherine; Ziemiecki, Andrew
CORPORATE SOURCE: Department of Clinical Research, University of Berne, Bern, CH-3004, Switz.
SOURCE: Journal of Cell Science (2002), 115(1), 25-37
CODEN: JNCSAI; ISSN: 0021-9533
PUBLISHER: Company of Biologists Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The authors have previously documented the cell-type-specific and hormone-dependent expression of the EphB4 receptor in the mouse mammary gland. To investigate its role in the biol. of the mammary gland, we have established transgenic mice bearing the EphB4 receptor under the control of the MMTV-LTR promoter, which represents the first transgenic mouse model to investigate the effect(s) of unscheduled expression of EphB4 in adult organisms. Transgene expression in the mammary epithelium was induced at puberty, increased during pregnancy, culminated at early lactation and persisted until day three of post-lactational involution. In contrast, expression of the endogenous EphB4 gene is downregulated during pregnancy, is essentially absent during lactation and is re-induced after day three of post-lactational involution. The unscheduled expression of EphB4 led to a delayed development of the mammary epithelium at puberty and during pregnancy. During pregnancy, less lobules were formed, these however exhibited more numerous but smaller alveolar units. Transgenic mammary glands were characterized by a fragile, irregular morphol. at lactation; however, sufficient functionality was maintained to nourish the young. Transgenic mammary glands exhibited untimely epithelial apoptotic cell death during pregnancy and abnormal epithelial DNA synthesis at early post-lactational involution, indicating a disturbed response to proliferative/apoptotic signals. Mammary tumors were not obsd. in the EphB4 transgenic animals; however, in double transgenic animals expressing both EphB4 and the neuT genes, tumor appearance was significantly accelerated and, in contrast to neuT-only animals, metastases were obsd. in the lung. These results implicate EphB4 in the regulation of tissue architecture, cellular growth response and establishment of the invasive phenotype in the adult mammary gland.
REFERENCE COUNT: 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS FORMAT RECORD. ALL CITATIONS AVAILABLE IN THE RE

L8 ANSWER 35 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:828415 CAPLUS
DOCUMENT NUMBER: 137:89412
TITLE: Detection of variations in the DNA methylation profile of genes in the determining the risk of disease
INVENTOR(S): Berlin, Kurt; Piepenbrock, Christian; Olek, Alexander
PATENT ASSIGNEE(S): Epigenomics A.-G., Germany
SOURCE: PCT Int. Appl., 636 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 68
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001077373	A2	20011018	WO 2001-XA1486	20010406
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, CF, CG, CI, CM, GA, GW, ML, MR, NE, SN, TD, TG				
DE 10019058	A1	20011220	DE 2000-10019058	20000406
WO 2001077373	A2	20011018	WO 2001-DE1486	20010406
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1274865	A2	20030115	EP 2001-953936	20010406
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

PRIORITY APPLN. INFO.: DE 2000-10019058 A 20000406
WO 2001-DE1486 W 20010406
DE 2000-10019173 A 20000407
DE 2000-10032529 A 20000630
DE 2000-10043826 A 20000901
WO 2001-EP3969 W 20010406

AB The invention relates to an oligonucleotide kit as probe for the detection of relevant variations in the DNA methylation of a target group of genes. The invention further relates to the use of the same for detg. the gene variant with regard to DNA methylation, a medical device, using an oligonucleotide kit, a method for detg. the methylation state of an individual and a method for the establishment of a model for establishing the probability of onset of a disease state in an individual. Such diseases may be: undesired pharmaceutical side-effects; cancerous diseases; CNS dysfunctions, injuries or diseases; aggressive symptoms or relational disturbances; clin., psychol. and social consequences of brain injury; psychotic disorders and personality disorders; dementia and/or assocd. syndromes; cardiovascular disease, dysfunction and damage; dysfunction, damage or disease of the gastrointestinal tract; dysfunction,

L8 ANSWER 35 OF 67 CAPLUS COPYRIGHT 2003 ACS (Continued)
damage or disease of the respiratory system; injury, inflammation, infection, immunity and/or anastasis; dysfunction, damage or disease of the body as an abnormal development process; dysfunction, damage or disease of the skin, muscle, connective tissue or bones; endocrine and metabolic dysfunction, damage or disease; headaches or sexual dysfunction.
This abstr. record is one of several records for this document necessitated by the large no. of index entries required to fully index the
document and publication system constraints.

L8 ANSWER 36 OF 67 CAPLUS COPYRIGHT 2003 ACS (Continued)
3.5 to 4.5 .mu.g/mL for EGFR-1, Tek-1, and EPGB4-1; and > 4.5 .mu.g/mL for
IGFR-1, AKT3-1, Met-1, Zap-1, Itk-1, FGFR1-1, and Fyn-1. I and compns. comprising them are useful for the treatment of disease or disease symptoms related to kinase inhibition, such as angiogenesis or **vasculogenesis** (no data).
REPERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 36 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:265404 CAPLUS
DOCUMENT NUMBER: 134:295842
TITLE: Preparation of triazine kinase inhibitors
INVENTOR(S): Armistead, David M.; Bemis, Jean E.; Buchanan, John L.; Dipietro, Lucian V.; Elbaum, Daniel; Habgood, Gregory J.; Kim, Joseph L.; Marshall, Teresa L.; Geuns-Meyer, Stephanie D.; Novak, Perry M.; Nunes, Joseph J.; Patel, Vinod F.; Toledo-Sherman, Leticia M.; Zhu, Xiaotian
PATENT ASSIGNEE(S): Kinetix Pharmaceuticals Inc., USA
SOURCE: PCT Int. Appl., 376 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001025220	A1	20010412	WO 2000-US27811	20001006
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1218360	A1	20020703	EP 2000-972036	20001006
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
JP 2003511378	T2	20030325	JP 2001-528166	20001006

PRIORITY APPLN. INFO.:
US 1999-158176P P 19991007
US 1999-166978P P 19991123
US 1999-170378P P 19991213
US 2000-183263P P 20000217
US 2000-215576P P 20000630
US 2000-219801P P 20000720
WO 2000-US27811 W 20001006

OTHER SOURCE(S): MARPAT 134:295842
AB Title triazine compds. (I) [wherein R1 and R2 = independently R3, R8, NHR3, NHR5, NHR6, NR5R5, NR5R6, SR5, SR6, SR3, OR5, OR6, OR3, COR3, or (un)substituted heterocyclyl or alkyl; R3 = independently aryl or (un)substituted Ph or heteroaryl; R5 = independently H, (un)substituted (cyclo)alkyl or alkenyl, alkynyl, cycloalkenyl, aryl, or haloalkyl; R6 = independently COR5, CO2R5, CONR5R5, C(NR5)NR5R5, or SOnR5; R8 = independently (un)substituted mono-, di-, or tricyclic ring system comprising 1-3, 1-6, or 1-9 heteroatoms, resp.; n = 1-2] were prepd. as inhibitors of enzymes that bind to ATP or GTP and/or catalyze phosphoryl transfer. For example, amination of 2,4-dichloro-1,3,5-triazine (prepn. given) with 3,4,5-trimethoxyaniline in DMF, followed by a second amination with 4-aminoveratrole in the presence of diisopropylethylamine in EtOH, yielded II. In kinase inhibition studies, II gave IC50 values of < 0.4 .mu.g/mL for KDR-1, PDGFRB-1, and Flt-1; 0.4 to 2.4 .mu.g/mL for Lck-1;

L8 ANSWER 37 OF 67 USPATFULL
ACCESSION NUMBER: 2001:130839 USPATFULL
TITLE: Cytokines that bind the cell surface receptor Hek
INVENTOR(S): Beckmann, M. Patricia, Poulsbo, WA, United States
Cerretti, Douglas P., Seattle, WA, United States
PATENT ASSIGNEE(S): Immunex Corporation, Seattle, WA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6274117	B1	20010814
APPLICATION INFO.:	US 1999-358734		19990721 (9)

RELATED APPLN. INFO.: Division of Ser. No. US 1998-57121, filed on 8 Apr 1998, now patented, Pat. No. US 5969110 Division of Ser. No. US 1995-453943, filed on 30 May 1995, now patented, Pat. No. US 5738844 Division of Ser. No. US 1994-240124, filed on 9 May 1994, now patented, Pat. No. US 5516658 Continuation-in-part of Ser. No. US 1993-161132, filed on 3 Dec 1993, now abandoned Continuation-in-part of Ser. No. US 1993-114426, filed on 30 Aug 1993, now abandoned Continuation-in-part of Ser. No. US 1993-109745, filed on 20 Aug 1993, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Mertz, Prema
LEGAL REPRESENTATIVE: Anderson, Kathryn A., Fowler, Kathleen
NUMBER OF CLAIMS: 7
EXEMPLARY CLAIM: 1
LINE COUNT: 1524
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Hek ligand (hek-L) polypeptides as well as DNA sequences, vectors and transformed host cells useful in providing hek-L polypeptides. The hek-L polypeptides bind to a cell surface receptor (hek) that is a member of the receptor tyrosine kinase family. Hek is expressed on cells that include certain tumor cell lines. The hek-L polypeptides also bind a distinct receptor tyrosine kinase known elk.

L8 ANSWER 38 OF 67 USPATFULL
ACCESSION NUMBER: 2001:75421 USPATFULL
TITLE: Methods of preventing and treating neurological disorders with compounds that modulate the function of the C-RET receptor protein tyrosine kinase
INVENTOR(S): Clary, Douglas, San Francisco, CA, United States
PATENT ASSIGNEE(S): Sugen, Inc., Redwood City, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6235769	B1	20010522
APPLICATION INFO.:	US 1998-109883		19980702 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1997-51715P	19970703 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Criares, Theodore J.
LEGAL REPRESENTATIVE: Foley & Lardner
NUMBER OF CLAIMS: 14
EXEMPLARY CLAIM: 1
LINE COUNT: 2371
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates in part to a method of preventing or treating an abnormal condition caused by an aberration in the function of the C-RET receptor, and specifically to the treatment and prevention of neurodegenerative disorders by administering a pharmaceutical composition that modulates the function of the C-RET receptor.

L8 ANSWER 39 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:354356 CAPLUS
DOCUMENT NUMBER: 135:120439
TITLE: Alterations of gene expression during colorectal carcinogenesis revealed by cDNA microarrays after laser-capture microdissection of tumor tissues and normal epithelia
AUTHOR(S): Kitahara, Osamu; Furukawa, Yoichi; Tanaka, Toshihiro; Kihara, Chikashi; Ono, Kenji; Yanagawa, Renpei; Nita, Marcelo E.; Takagi, Toshihisa; Nakamura, Yusuke; Tsunoda, Tatsuhiko
CORPORATE SOURCE: Human Genome Center, Institute of Medical Science, The University of Tokyo, Tokyo, 108-8639, Japan
SOURCE: Cancer Research (2001), 61(9), 3544-3549
CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English
AB To identify a set of genes involved in the development of colorectal carcinogenesis, we compared expression profiles of colorectal cancer cells from eight tumors with corresponding noncancerous colonic epithelia using a DNA microarray consisting of 9216 human genes. These cell populations had been rendered homogeneous by laser-capture microdissection. Expression change in more than half of the tumors was obsd. for 235 genes, i.e., 44 up-regulated and 191 down-regulated genes. The differentially expressed genes include those assocd. with signal transduction, metabolizing enzymes, prodn. of reactive oxygen species, cell cycle, transcription, mitosis, and apoptosis. Subsequent examn. of 10 genes (five up-regulated and five down-regulated) by semiquant. reverse transcription-PCR using the eight tumors together with an addnl. 12 samples substantiated the reliability of our anal. The extensive list of genes identified in these expts. provides a large body of potentially valuable information of colorectal carcinogenesis and represents a source of novel targets for cancer therapy.
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 40 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:621393 CAPLUS
DOCUMENT NUMBER: 136:115945
TITLE: Differential gene expression during capillary morphogenesis in 3D collagen matrices: regulated expression of genes involved in basement membrane matrix assembly, cell cycle progression, cellular differentiation and G-protein signaling
AUTHOR(S): Bell, Scott E.; Mavila, Anil; Salazar, Rene; Bayless, Kayla J.; Kanagala, Suhasini; Maxwell, Steven A.; Davis, George E.
CORPORATE SOURCE: Department of Pathology and Laboratory Medicine, Texas A and M University System Health Science Center, College Station, TX, 77843-1114, USA
SOURCE: Journal of Cell Science (2001), 114(15), 2755-2773
CODEN: JNCSAI; ISSN: 0021-9533
PUBLISHER: Company of Biologists Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We have performed a screening anal. of differential gene expression using a defined in vitro model of human capillary tube formation. Gene array, differential display and cDNA library screening were used to identify both known and novel differentially expressed genes. Major findings include: the upregulation and functional importance of genes assocd. with basement membrane matrix assembly; the upregulation of growth factors, transcription factors, anti-apoptotic factors, markers of endothelial cell differentiation, JAK-STAT signalling mols., adhesion receptors, proteinase inhibitors and actin regulatory proteins; and expression changes consistent with inhibition of cell cycle progression, increased cholesterol biosynthesis, decreased ubiquitin-proteasome mediated degrdn., and activation of G-protein signaling pathways. Using DNA microarray anal., the most induced genes at 8, 24 and 48 h compared with those at 0 h were jagged-1, stanniocalcin and angiopoietin-2, whereas the most repressed genes were connective tissue growth factor, fibulin-3 and RGS-5. In addn., the full length coding sequence of two novel regulated capillary morphogenesis genes (CMGs) are presented. CMG-1 encodes a predicted intracellular 65 kDa protein with coiled-coil domains. A CMG-1-green fluorescent protein (GFP) chimera was obsd. to target to an intracellular vesicular compartment. A second novel gene, CMG-2, was found to encode a predicted intracellular protein of 45 kDa contg. a transmembrane segment and a CMG-2-GFP chimera was obsd. to target to the endoplasmic reticulum. A recombinant portion of CMG-2 was found to bind collagen type IV and laminin, suggesting a potential role in basement membrane matrix synthesis and assembly. These data further elucidate the genetic events regulating capillary tube formation in a 3D matrix environment.
REFERENCE COUNT: 125 THERE ARE 125 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 41 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:613123 CAPLUS
DOCUMENT NUMBER: 135:286338
TITLE: Stromal cells expressing ephrin-B2 promote the growth and sprouting of ephrin-B2+ endothelial cells
AUTHOR(S): Zhang, Xiu-Qin; Takakura, Nobuyuki; Oike, Yuichi; Inada, Tomohisa; Gale, Nicholas W.; Yancopoulos, George D.; Suda, Toshio
CORPORATE SOURCE: Department of Cell Differentiation, Institute of Molecular Embryology and Genetics, Kumamoto University, Kumamoto, 860-0811, Japan
SOURCE: Blood (2001), 98(4), 1028-1037
CODEN: BLOOAW; ISSN: 0006-4971
PUBLISHER: American Society of Hematology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Ephrin-B2 is a transmembrane ligand that is specifically expressed on arterial endothelial cells (ECs) and surrounding cells and interacts with multiple EphB class receptors. Conversely, EphB4, a specific receptor for ephrin-B2, is expressed on venous ECs, and both ephrin-B2 and EphB4 play essential roles in vascular development. The bidirectional signals between EphB4 and ephrin-B2 are thought to be specific for the interaction between arteries and veins and to regulate cell mixing and the making of particular boundaries. However, the mol. mechanism during vasculogenesis and angiogenesis remains unclear. Manipulative functional studies were performed on these proteins in an endothelial cell system. Using in vitro stromal cells (OP9 cells) and a paraaortic spianchnopleura (P-Sp) coculture system, these studies found that the stromal cells expressing ephrin-B2 promoted vascular network formation and ephrin-B2+ EC proliferation and that they also induced the recruitment and proliferation of .alpha.-smooth muscle actin (.alpha.-SMA)-pos. cells. Stromal cells expressing EphB4 inhibited vascular network formation, ephrin-B2+ EC. proliferation, and .alpha.-SMA+ cell recruitment and proliferation. Thus, these data suggest that ephrin-B2 and EphB4 mediate reciprocal interactions between arterial and venous ECs and surrounding cells to form each characteristic vessel.
REFERENCE COUNT: 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 42 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:111343 CAPLUS
DOCUMENT NUMBER: 136:214603
TITLE: Gene expression profiling for molecular identification of high-risk breast cancer patients
AUTHOR(S): Ahr, A.; Holtrich, U.; Solbach, C.; Scharl, A.; Strebhardt, K.; Karn, T.; Kaufmann, M.
CORPORATE SOURCE: Bereich "Molekulare Gynakologie und Geburtshilfe", Universitats-Frauenklinik Frankfurt, Frankfurt, 60590, Germany
SOURCE: Geburtshilfe und Frauenheilkunde (2001), 61(12), 954-963
CODEN: GEFRA2; ISSN: 0016-5751
PUBLISHER: Georg Thieme Verlag
DOCUMENT TYPE: Journal
LANGUAGE: German
AB Many **tumors** are subclassified with genetic markers to improve therapy and follow-up. A goal is to use DNA arrays as a tool to understand and classify **tumors** on the basis of gene expression patterns. Global detn. of cellular transcriptional activity may identify gene expression signatures that predict the clin. behavior of **tumors**. We performed DNA array studies and subsequent cluster analyses to establish a mol. profile of 73 breast cancer specimens (T1-4, N0-1, M0-1). Our analyses identified a group of transcriptionally related specimens. 14 Of these 16 **tumors** were node pos. This group had an accumulation of patients with distant metastases at the time of diagnosis (25% vs. 4%). Differentially expressed marker genes in conjunction with sample clustering algorithms provide a novel mol. classification system for breast cancers. This may help identify patients at high risk of recurrence.
REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 43 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:340158 CAPLUS
DOCUMENT NUMBER: 135:301859
TITLE: Expression of receptor tyrosine kinase EphB4 and its ligand ephrin-B2 is associated with malignant potential in endometrial cancer
AUTHOR(S): Takai, Noriyuki; Miyazaki, Tami; Fujisawa, Kayo; Nasu, Kaei; Miyakawa, Isao
CORPORATE SOURCE: Department of Obstetrics and Gynecology, Oita Medical University, Oita, 879-5593, Japan
SOURCE: Oncology Reports (2001), 8(3), 567-573
CODEN: OCRPEW; ISSN: 1021-335X
PUBLISHER: Oncology Reports
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The protein kinases includes many oncogenes and growth-factor receptors, as well as genes that are involved in cell cycle regulation. EphB4 receptors are a subfamily of receptor tyrosine kinases that are activated by ephrin-B2 ligands and are thought to play an important role in the development and oncogenesis of various tissues. However, very little exptl. evidence exists to support this hypothesis. To elucidate the involvement of EphB4 and ephrin-B2 in endometrial carcinogenesis, we used fluorescent immunohistochem. to analyze serial frozen sections of 20 endometrial carcinomas and 20 normal endometria for EphB4 and ephrin-B2 protein expression. We analyzed the relationship between the patient's characteristics and the percentages of EphB4- and ephrin-B2-stained cells.
EphB4 expression was significantly assocd. with histol. grade ($p<0.001$) and certain clin. stages. Ephrin-B2 Expression was significantly assocd. with the presence of invasion to >1/2 myometrium ($p=0.002$). Our results demonstrate that increased EphB4 and ephrin-B2 expression may reflect or induce in endometrial carcinomas increased potential for growth and **tumorigenicity**. Furthermore, these results suggest that EphB4 receptor kinase may modulate the biol. behavior of endometrial carcinomas through autocrine and/or paracrine activation, which is caused by ephrin-B2 ligands that are expressed in the same or neighboring cells by immunohistochem.
REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 44 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:75014 CAPLUS
DOCUMENT NUMBER: 134:247522
TITLE: Expression of EphrinB2 Identifies a Stable Genetic Difference Between Arterial and Venous **Vascular** Smooth Muscle as Well as Endothelial Cells, and Marks Subsets of Microvessels at Sites of Adult Neovascularization
AUTHOR(S): Shin, Donghun; Garcia-Cardena, Guillermo; Hayashi, Shin-Ichiro; Gerety, Sebastian; Asahara, Takayuki; Stavrakis, George; Isner, Jeffrey; Folkman, Judah; Gimbrone, Michael A., Jr.; Anderson, David J.
CORPORATE SOURCE: Division of Biology 216-76, California Institute of Technology, Pasadena, CA, USA
SOURCE: Developmental Biology (Orlando, FL, United States) (2001), 230(2), 139-150
CODEN: DEBIAO; ISSN: 0012-1606
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The transmembrane ligand ephrinB2 and its receptor tyrosine kinase EphB4 are mol. markers of embryonic arterial and venous endothelial cells, resp., and are essential for angiogenesis. Here we show that expression of ephrinB2 persists in adult arteries where it extends into some of the smallest diam. microvessels, challenging the classical view that capillaries have neither arterial nor venous identity. EphrinB2 also identifies arterial microvessels in several settings of adult neovascularization, including tumor angiogenesis, contravening the dogma that tumor vessels arise exclusively from postcapillary venules. Unexpectedly, expression of ephrinB2 also defines a stable genetic difference between arterial and venous **vascular** smooth muscle cells. These observations argue for revisions of classical concepts of capillary identity and the topog. of neovascularization. They also imply that ephrinB2 may be functionally important in neovascularization and in arterial smooth muscle, as well as in embryonic angiogenesis. (c) 2001 Academic Press.
REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 45 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:267908 CAPLUS
DOCUMENT NUMBER: 134:361746
TITLE: The cytoplasmic domain of the ligand ephrinB2 is required for **vascular** morphogenesis but not cranial neural crest migration
AUTHOR(S): Adams, Ralf H.; Diella, Francesca; Hennig, Silvia; Helmbacher, Francoise; Deutsch, Urban; Klein, Rudiger
CORPORATE SOURCE: European Molecular Biology Laboratory, Heidelberg, D-69117, Germany
SOURCE: Cell (Cambridge, MA, United States) (2001), 104(1), 57-69
CODEN: CELLB5; ISSN: 0092-8674
PUBLISHER: Cell Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The transmembrane ligand ephrinB2 and its cognate Eph receptor tyrosine kinases are important regulators of **vascular** morphogenesis. EphrinB2 may have an active signaling role, resulting in bi-directional signal transduction downstream of both ephrinB2 and Eph receptors. To sep. the ligand and receptor-like functions of ephrinB2 in mice, we replaced the endogenous gene by cDNAs encoding either carboxy-terminally truncated (ephrinB2.DELTA.C) or, as a control, full-length ligand (ephrinB2WT). While homozygous ephrinB2WT/WT animals were viable and fertile, loss of the ephrinB2 cytoplasmic domain resulted in midgestation lethality similar to ephrinB2 null mutants (ephrinB2KO). The truncated ligand was sufficient to restore guidance of migrating cranial neural crest cells, but ephrinB2.DELTA.C/.DELTA.C embryos showed defects in **vasculogenesis** and angiogenesis very similar to those obsd. in ephrinB2KO/KO animals. Our results indicate distinct requirements of functions mediated by the ephrinB carboxy-terminus for developmental processes in the vertebrate embryo.
REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 46 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:131595 CAPLUS
DOCUMENT NUMBER: 137:76792
TITLE: Receptor protein tyrosine kinase EphB4 is up-regulated in colon cancer
AUTHOR(S): Stephenson, Sally-Anne; Slomka, Stefan; Douglas, Evelyn L.; Hewett, Peter J.; Hardingham, Jennifer E.
CORPORATE SOURCE: Dep. Hematology, The Queen Elizabeth Hospital, Woodville, Australia
SOURCE: BMC Molecular Biology [online computer file] (2001), 2, No pp. given
CODEN: BMBMC4; ISSN: 1471-2199
URL: http://www.biomedcentral.com/1471-2199/2/15
PUBLISHER: BioMed Central Ltd.
DOCUMENT TYPE: Journal; (online computer file)
LANGUAGE: English
AB Background: We have used com. cDNA arrays to identify EphB4 as a gene that is up-regulated in colon cancer tissue when compared with matched normal tissue from the same patient. Results: Quant. RT-PCR anal. of the expression of the EphB4 gene has shown that its expression is increased in 82% of tumor samples when compared with the matched normal tissue from the same patient. Using immunohistochem. and Western anal. techniques with an EphB4-specific antibody, we also show that this receptor is expressed in the epithelial cells of the tumor tissue and either not at all, or in only low levels, in the normal tissue. Conclusion: The results presented here supports the emerging idea the Eph receptors play a role in tumor formation and suggests that further elucidation of this signaling pathway may identify useful targets for cancer treatment therapies.
REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE
FORMAT

L8 ANSWER 47 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:368136 CAPLUS
DOCUMENT NUMBER: 133:808
TITLE: Uses for Eph receptor antagonists and agonists to treat vascular disorders
INVENTOR(S): Aguet, Michel
PATENT ASSIGNEE(S): Genentech, Inc., USA
SOURCE: PCT Int. Appl., 46 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000030673	A1	20000602	WO 1999-US27534	19991118
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1135153	A1	20010926	EP 1999-960524	19991118
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002530350	T2	20020917	JP 2000-583556	19991118

PRIORITY APPLN. INFO.: US 1998-109275P P 19981120
WO 1999-US27534 W 19991118
AB The present application describes methods of inhibiting or stimulating angiogenesis in a mammal comprising administering to the mammal an effective amt. of an Eph receptor antagonist or agonist, resp. Articles of manuf. for use in relation to these methods are also described.
REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE
FORMAT

L8 ANSWER 48 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:314864 CAPLUS
DOCUMENT NUMBER: 132:344076
TITLE: Method for detecting endocrine disruptor-responsive genes and for screening endocrine disruptors
INVENTOR(S): Kondo, Akihiro; Sagawa, Hiroaki; Mineno, Junichi; Kimizuka, Fusao; Kato, Ikunoshin
PATENT ASSIGNEE(S): Takara Shuzo Co., Ltd., Japan
SOURCE: PCT Int. Appl., 81 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000026404	A1	20000511	WO 1999-JP5964	19991028
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9964878	A1	20000522	AU 1999-64878	19991028
EP 1126035	A1	20010822	EP 1999-952794	19991028
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.: JP 1998-310285 A 19981030
WO 1999-JP5964 W 19991028
AB A method and compns. for detecting genes affected by endocrine-disrupting chems. and for identifying endocrine-disrupting chems. are claimed. The method comprises prepg. a nucleic acid sample contg. mRNAs or cDNAs originating in cells, tissues, or organisms which have been brought into contact with a sample contg. the endocrine disruptor. The nucleic acid sample is hybridized with DNA arrays wherein genes which might be affected by the endocrine disruptor or DNA fragments originating in these genes have been fixed. The results obtained are then compared with the results obtained with the control sample to select the gene affected by the endocrine disruptor. Endocrine disruptors are selected from dioxins, org. chloro compds., phenols, futalic acid esters, arom. hydrocarbons, agrochems., org. tin compds., and estrogens, among others. The effect of 3 chems., 17-.beta. estradiol (E2), diethylstilbestrol (DES), and bisphenol A (BisA) on 33 candidate genes belonging to the categories of nuclear receptor/nuclear receptor transcriptional coupling, kinase-type signal transducer, gonad differentiation factor, oncogene, and receptor-type kinase, were examd. by the method of this invention. Expression of most of the genes was either increased or decreased by exposure to these chems.
REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE
FORMAT

L8 ANSWER 49 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:722227 CAPLUS
DOCUMENT NUMBER: 134:3346
TITLE: Implications of EPHB6, EFNB2, and EFNB3 expressions in human neuroblastoma
AUTHOR(S): Tang, Xao X.; Zhao, Huaqing; Robinson, Marjorie E.; Cohen, Brian; Cnaan, Avital; London, Wendy; Cohn, Susan L.; Cheung, Nai-Kong V.; Brodeur, Garrett M.; Evans, Audrey E.; Ikegaki, Naohiko
CORPORATE SOURCE: Division of Oncology, The Children's Hospital of Philadelphia, Philadelphia, PA, 19104-4318, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2000), 97(20), 10936-10941
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Neuroblastoma (NB) is a common pediatric tumor that exhibits a wide range of biol. and clin. heterogeneity. EPH (erythropoietin-producing hepatoma amplified sequence) family receptor tyrosine kinases and ligand ephrins play pivotal roles in neural and cardiovascular development. High-level expression of transcripts encoding EPHB6 receptors (EPHB6) and its ligands ephrin-B2 and ephrin-B3 (EFNB2, EFNB3) is assocd. with low-stage NB (stages 1, 2, and 4S) and high TrkA expression. In this study, we showed that EFNB2 and TrkA expressions were assocd. with both tumor stage and age, whereas EPHB6 and EFNB3 expressions were solely assocd. with tumor stage, suggesting that these genes were expressed in distinct subsets of NB. Kaplan-Meier and Cox regression analyses revealed that high-level expression of EPHB6, EFNB2, and EFNB3 predicted favorable NB outcome ($P < 0.005$), and their expression combined with TrkA expression predicted the disease outcome more accurately than each variable alone ($P < 0.00005$). Interestingly, if any one of the four genes (EPHB6, EFNB2, EFNB3, or TrkA) was expressed at high levels in NB, the patient survival was excellent (>90%). To address whether a good disease outcome of NB was a consequence of high-level expression of a "favorable NB gene," we examd. the effect of EPHB6 on NB cell lines. Transfection of EPHB6 cDNA into IMR5 and SY5Y expressing little endogenous EPHB6 resulted in inhibition of their clonogenicity in culture. Furthermore, transfection of EPHB6 suppressed the tumorigenicity of SY5Y in a mouse xenograft model, demonstrating that high-level expressions of favorable NB genes, such as EPHB6, can in fact suppress malignant phenotype of unfavorable NB.
REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE
FORMAT

L8 ANSWER 50 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:395628 CAPLUS
DOCUMENT NUMBER: 133:130254
TITLE: Expression of Eph receptors and ephrins is differentially regulated by E-cadherin
AUTHOR(S): Orsulic, Sandra; Kemler, Rolf
CORPORATE SOURCE: Max-Planck-Institut fur Immunbiologie, Freiburg, D-79108, Germany
SOURCE: Journal of Cell Science (2000), 113(10), 1793-1802
CODEN: JNCSAI; ISSN: 0021-9533
PUBLISHER: Company of Biologists Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB E-cadherin is the main cell adhesion mol. of early embryonic and adult epithelial cells. Downregulation of E-cadherin is assocd. with epithelial-mesenchymal transition during embryonic mesoderm formation and tumor progression. To identify genes whose expression is affected by the loss of E-cadherin, we compared mRNA expression patterns between wild-type and E-cadherin null mutant embryonic stem (ES) cells. We found that expression of several Eph receptors and ephrins is dependent on E-cadherin. Rescue of E-cadherin null ES cells with E-cadherin cDNA restores the wild-type expression pattern of Eph family members. Rescue of E-cadherin null ES cells with N-cadherin cDNA does not restore the wild-type expression pattern, indicating that the regulation of differential expression of Eph family members is specific to E-cadherin. Constitutive ectopic expression of E-cadherin in non-epithelial NIH3T3 cells results in the prodn. of the EphA2 receptor. In epithelial cells, E-cadherin is required for EphA2 receptor localization at cell-cell contacts; in the absence of functional E-cadherin, EphA2 localizes to the perinuclear region. Our results indicate that E-cadherin may be directly or indirectly required for the membrane localization of Eph receptors and their membrane-bound ligands.

REFERENCE COUNT: 87 THERE ARE 87 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 51 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:147875 CAPLUS
DOCUMENT NUMBER: 132:248718
TITLE: The receptor tyrosine kinase EphB4 and ephrin-B ligands restrict angiogenic growth of embryonic veins in Xenopus laevis
AUTHOR(S): Helbling, Paul M.; Saulnier, Didier M. E.; Brandli, Andre W.
CORPORATE SOURCE: Institute of Cell Biology, Swiss Federal Institute of Technology, Zurich, CH-8093, Switz.
SOURCE: Development (Cambridge, United Kingdom) (2000), 127(2), 269-278
CODEN: DEVPED; ISSN: 0950-1991
PUBLISHER: Company of Biologists Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We investigated the role of EphB receptors and their ligands during embryonic blood vessel development in X. laevis. In a survey of tadpole-stage Xenopus embryos for EphB receptor expression, we detected expression of EphB4 receptors in the posterior cardinal veins and their derivs., the intersomitic veins. Vascular expression of other EphB receptors, including EphB1, EphB2, or EphB3, could however not be obsd., suggesting that EphB4 is the principal EphB receptor of the early embryonic vasculature of Xenopus. Furthermore, we found that ephrin-B ligands are expressed complementary to EphB4 in the somites adjacent to the migratory pathways taken by intersomitic veins during angiogenic growth. We performed RNA injection expts. to study the function of EphB4 and its ligands in intersomitic vein development. Disruption of EphB4 signaling by dominant neg. EphB4 receptors or misexpression of ephrin-B ligands in Xenopus embryos resulted in intersomitic veins growing abnormally into the adjacent somitic tissue. Our findings demonstrate that EphB4 and B-class ephrins act as regulators of angiogenesis possibly by mediating repulsive guidance cues to migrating endothelial cells.

REFERENCE COUNT: 68 THERE ARE 68 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 52 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:795994 CAPLUS
DOCUMENT NUMBER: 132:31744
TITLE: Gene probes used for genetic profiling in healthcare screening and planning
INVENTOR(S): Roberts, Gareth Wyn
PATENT ASSIGNEE(S): Genostic Pharma Ltd., UK
SOURCE: PCT Int. Appl., 745 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9964627	A2	19991216	WO 1999-GB1780	19990604

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:

GB 1998-12099	A	19980606
GB 1998-13291	A	19980620
GB 1998-13611	A	19980624
GB 1998-13835	A	19980627
GB 1998-14110	A	19980701
GB 1998-14580	A	19980707
GB 1998-15438	A	19980716
GB 1998-15574	A	19980718
GB 1998-15576	A	19980718
GB 1998-16085	A	19980724
GB 1998-16086	A	19980724
GB 1998-16921	A	19980805
GB 1998-17097	A	19980807
GB 1998-17200	A	19980808
GB 1998-17632	A	19980814
GB 1998-17943	A	19980819

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response.

In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the no. of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide crit. clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of

L8 ANSWER 52 OF 67 CAPLUS COPYRIGHT 2003 ACS (Continued)

genes enables the invention of a design for genetic profiling technologies which comprises of the identification of the core group of genes and their sequence variants required to provide a broad base of clin. prognostic information - "genostics". The "Genostic" profiling of patients and persons will radically enhance the ability of clinicians, healthcare professionals and other parties to plan and manage healthcare provision and the targeting of appropriate healthcare resources to those deemed most in need. The use of this invention could also lead to a host of new applications for such profiling technologies, such as identification of persons with particular work or environment related risk, selection of applicants for employment, training or specific opportunities or for the enhancing of the planning and organization of health services, education services and social services.

L8 ANSWER 53 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:795993 CAPLUS
DOCUMENT NUMBER: 132:31743
TITLE: Gene probes used for genetic profiling in healthcare screening and planning
INVENTOR(S): Roberts, Gareth Wyn
PATENT ASSIGNEE(S): Genostic Pharma Limited, UK
SOURCE: PCT Int. Appl., 149 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9964626	A2	19991216	WO 1999-GB1779	19990604
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2330929	AA	19991216	CA 1999-2330929	19990604
AU 9941586	A1	19991230	AU 1999-41586	19990604
AU 9941587	A1	19991230	AU 1999-41587	19990604
GB 2339200	A1	20000119	GB 1999-12914	19990604
GB 2339200	B2	20010912		
EP 1084273	A1	20010321	EP 1999-925207	19990604
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.:

GB 1998-12098	A	19980606
GB 1998-28289	A	19981223
GB 1998-16086	A	19980724
GB 1998-16921	A	19980805
GB 1998-17097	A	19980807
GB 1998-17200	A	19980808
GB 1998-17632	A	19980814
GB 1998-17943	A	19980819
WO 1999-GB1779	W	19990604

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response.
In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the no. of genes and

L8 ANSWER 53 OF 67 CAPLUS COPYRIGHT 2003 ACS (Continued)
their configurations (mutations and polymorphisms) needed to be identified in order to provide crit. clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies.

L8 ANSWER 54 OF 67 USPATFULL
ACCESSION NUMBER: 1999:128726 USPATFULL
TITLE: Antibodies that bind hek ligands
INVENTOR(S): Beckmann, M. Patricia, Poulsbo, WA, United States
Cerretti, Douglas P., Seattle, WA, United States
PATENT ASSIGNEE(S): Immunex Corporation, Seattle, WA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5969110		19991019
APPLICATION INFO.:	US 1998-57121		19980408 (9)

RELATED APPLN. INFO.: Division of Ser. No. US 1995-453943, filed on 30 May 1995, now patented, Pat. No. US 5738844 which is a division of Ser. No. US 1994-240124, filed on 9 May 1994, now patented, Pat. No. US 5516658 which is a continuation-in-part of Ser. No. US 1993-161132, filed on 3 Dec 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-114426, filed on 30 Aug 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-109745, filed on 20 Aug 1993, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Mertz, Prema
LEGAL REPRESENTATIVE: Anderson, Kathryn A.
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
LINE COUNT: 1771
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Antibodies specific for a hek-L may be generated, using a hek-L polypeptide or fragment thereof as an immunogen. The antibodies may be monoclonal.

L8 ANSWER 55 OF 67 USPATFULL
ACCESSION NUMBER: 1999:75761 USPATFULL
TITLE: Cytokine designated LERK-6
INVENTOR(S): Cerretti, Douglas P., Seattle, WA, United States
PATENT ASSIGNEE(S): Immunex Corporation, Seattle, WA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5919905		19990706
APPLICATION INFO.:	US 1997-920440		19970829 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1995-538709, filed on 3 Oct 1995 which is a continuation-in-part of Ser. No. US 1994-318393, filed on 5 Oct 1994, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Draper, Garnette D.
LEGAL REPRESENTATIVE: Henry, Janis C.
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
LINE COUNT: 1560
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention is directed to LERK-6 as a purified and isolated protein, the DNA encoding the LERK-6, host cells transfected with cDNAs encoding LERK-6.

L8 ANSWER 56 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:427998 CAPLUS
DOCUMENT NUMBER: 131:241175
TITLE: High-level expression of EPHB6, EFNB2, and EFNB3 is associated with low tumor stage and high TrkA expression in human neuroblastomas
AUTHOR(S): Tang, Xao X.; Evans, Audrey E.; Zhao, Huaqing; Cnaan, Avital; London, Wendy; Cohn, Susan L.; Brodeur, Garrett M.; Ikegaki, Naohiko
CORPORATE SOURCE: Divisions of Oncology, The Children's Hospital of Philadelphia, Abramson Research Center, Philadelphia, PA, 19104-4318, USA
SOURCE: Clinical Cancer Research (1999), 5(6), 1491-1496
CODEN: CCREF4; ISSN: 1078-0432
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Neuroblastoma (NB) is a common pediatric tumor of neural crest origin that is biol. and clin. heterogeneous. EPH family receptor tyrosine kinases and ephrin ligands play fundamental roles in neuro-developmental processes. Recently, we found that NB cell lines expressed several EPHB and EFNB transcripts, which encode EPHB subgroup receptors and ephrin-B subgroup ligands, resp. To explore the role of EPHB receptors and ephrin-B ligands in the biol. of NB, we examd. the expression of EPHB and EFNB transcripts in 47 primary NB specimens. Multiple EPHB and EFNB transcripts were expressed in all of the NB tumors examd., suggesting the involvement of these transcripts in modulating the biol. behavior of NB. Higher levels of EPHB6, EFNB2, and EFNB3 expression were found in low-stage tumors (stage 1, 2, and 4S) than in advanced-stage tumors (stage 3 and 4; P = 0.0013, P = 0.0048, and P = 0.027, resp.). Expression of TrkA, a well-established prognostic marker of favorable NB, was pos. correlated with EPHB6, EFNB2, and EFNB3 expression (P < 0.0001, P = 0.0019, and P = 0.0001, resp.). MYCN-amplified tumors expressed lower levels of EPHB6, EFNB2, EFNB3, and TrkA transcripts compared to nonamplified tumors (P = 0.0006, P = 0.0023, P = 0.0048, and P = 0.0001, resp.). These data suggest that high-level expression of EPHB6, EFNB2, and EFNB3 is assocd. with favorable NB and that low-level expression of EPHB6, EFNB2, and EFNB3 correlates with aggressive MYCN-amplified NB. Thus, EPHB6, EFNB2, and EFNB3 may have biol. relevance in NB. Further investigation on the biol. of these genes may help provide insight into the treatment of NB.
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 57 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:141870 CAPLUS
DOCUMENT NUMBER: 131:3510
TITLE: Coexpression of transcripts encoding EPHB receptor protein tyrosine kinases and their ephrin-B ligands in human small cell lung carcinoma
AUTHOR(S): Tang, Xao X.; Brodeur, Garrett M.; Campling, Barbara G.; Ikegaki, Naohiko
CORPORATE SOURCE: Division of Oncology, The Children's Hospital of Philadelphia, Philadelphia, PA, 19104, USA
SOURCE: Clinical Cancer Research (1999), 5(2), 455-460
CODEN: CCREF4; ISSN: 1078-0432
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The EPH family is the largest subfamily of receptor protein tyrosine kinases, consisting of the EPHA and EPHB subgroups. Ephrin-B1, ephrin-B2, and ephrin-B3 are ligands of the EPHB subgroup and are encoded by the EFNB1, EFNB2, and EFNB3 genes, resp. We have shown previously that EPHB2 transcripts are expressed in six small cell lung carcinoma (SCLC) cell lines. In this study, we examd. the expression of EPHB1, EPHB2, EPHB3, EPHB4, and EPHB6 in 4 SCLC tumor specimens and 14 cell lines including 3 cell lines derived from these tumor specimens. To investigate whether potential autocrine loops of EPHB receptors and ephrin-B ligands exist in SCLC, the expression of EFNB1, EFNB2, and EFNB3 was also examd. Our data show that transcripts encoding multiple members of the EPHB subgroup and the ephrin-B subgroup are coexpressed in SCLC cell lines and tumors. These results suggest that the EPHB subgroup receptor kinases may modulate the biol. behavior of SCLC through autocrine and/or juxtacrine activation by ephrin-B ligands that are expressed in the same or neighboring cells.
REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 58 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:602740 CAPLUS
DOCUMENT NUMBER: 131:319486
TITLE: Characterization of ephrin-A1 and ephrin-A4 as ligands for the ephA8 receptor protein tyrosine kinase
AUTHOR(S): Choi, Sunga; Jeong, Jaemin; Kim, Taewoong; Park, Soochul
CORPORATE SOURCE: Institute of Environment and Life Science, Hallym University, Chuncheon, 200-702, S. Korea
SOURCE: Molecules and Cells (1999), 9(4), 440-445
CODEN: MOCEEK; ISSN: 1016-8478
PUBLISHER: Springer-Verlag Singapore Pte. Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The Eph receptors are the largest known family of receptor protein tyrosine kinases, which play important roles with their ligands called ephrin in the neural development, angiogenesis, and vascular network assembly. It was previously shown that ephrin-A2, -A3 and -A5 bind to, and activate the EphA8 receptor tyrosine kinase, resp. In this study, we have examd. if there are other addnl. ephrin ligands interacting with the EphA8 receptor tyrosine kinase expressed in NIH3T3 fibroblasts. For this purpose, we have constructed chimeric ephrin-A1, -A4, -B1, -B2 or -B3 ligands consisting of the Fc portion of human IgG fused to their carboxyl-terminus. Both ephrin-A1 and ephrin-A4 chimeric ligands efficiently bound to the EphA8 receptor expressed in NIH3T3 fibroblasts, whereas the transmembrane ligands including ephrin-B1, -B2 and -B3 did not. Addnl. we have demonstrated that both the EphA8-TrkB chimeric receptor and the EphA8 receptor expressed in NIH3T3 fibroblasts are efficiently tyrosine-phosphorylated upon stimulating with ephrin-A1 or -A4 but none of transmembrane ephrin-B proteins. These results strongly indicate that the EphA8 receptor functions exclusively as an glycosyl phosphatidylinositol (GPI)-linked ephrin ligand-dependent receptor protein tyrosine kinase.
REFERENCE COUNT: 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 59 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:644221 CAPLUS
DOCUMENT NUMBER: 131:334941
TITLE: Symmetrical mutant phenotypes of the receptor EphB4 and its specific transmembrane ligand ephrin-B2 in cardiovascular development
AUTHOR(S): Gerety, Sebastian S.; Wang, Hai U.; Chen, Zhou-Feng; Anderson, David J.
CORPORATE SOURCE: Division of Biology Howard Hughes Medical Institute, California Institute of Technology, Pasadena, CA, 91125, USA
SOURCE: Molecular Cell (1999), 4(3), 403-414
CODEN: MOCEFL; ISSN: 1097-2765
PUBLISHER: Cell Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Ephrin-B2 is a transmembrane ligand that is specifically expressed on arteries but not veins and that is essential for cardiovascular development. However, ephrin-B2 is also expressed in nonvascular tissues and interacts with multiple EphB class receptors expressed in both endothelial and nonendothelial cell types. Thus, the identity of the relevant receptor for ephrin-B2 and the site(s) where these mols. interact to control angiogenesis were not clear. Here we show that EphB4, a specific receptor for ephrin-B2, is exclusively expressed by vascular endothelial cells in embryos and is preferentially expressed on veins. A targeted mutation in EphB4 essentially phenocopies the mutation in ephrin-B2. These data indicate that ephrin-B2-EphB4 interactions are intrinsically required in vascular endothelial cells and are consistent with the idea that they mediate bidirectional signaling essential for angiogenesis.
REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 60 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:24291 CAPLUS
DOCUMENT NUMBER: 132:178235
TITLE: Comparative analysis of embryonic gene expression defines potential interaction sites for Xenopus EphB4 receptors with ephrin-B ligands
AUTHOR(S): Helbling, Paul M.; Saulnier, Didier M. E.; Robinson, Vicky; Christiansen, Jeff H.; Wilkinson, David G.; Brandli, Andre W.
CORPORATE SOURCE: Institute of Cell Biology, Swiss Federal Institute of Technology, Zurich, CH-8093, Switz.
SOURCE: Developmental Dynamics (1999), 216(4/5), 361-373
CODEN: DEDYEI; ISSN: 1058-8388
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The Eph family of receptor tyrosine kinases and their ligands, the ephrins, act as signaling mols. regulating the migratory behavior of neurons and neural crest cells, and are implicated in tissue patterning, blood vessel formation, and tumorigenesis. On the basis of structural similarities and overlapping binding specificities, Eph receptors as well as their ligands can be divided into A and B subfamilies with orthologues found in all vertebrates. We describe here the isolation of cDNAs encoding Xenopus EphB4 receptors and show that embryonic expression is prominently assocd. with the developing **vasculature**, newly forming somites, the visceral arches, and non-neuronal tissues of the embryonic head. In a screen to identify potential ligands for EphB4 in Xenopus embryos, we isolated cDNAs for the Xenopus ephrin-B2 and -B3, which demonstrates that the Xenopus genome harbors genes encoding orthologues to all 3 currently known mammalian ephrin-B genes. We next performed in situ hybridizations to identify tissues and organs where EphB4 receptors may encounter ephrin-B ligands during embryonic development. Our anal. revealed distinct, but overlapping patterns of ephrin-B gene expression. Interestingly, each ephrin-B ligand displayed expression domains either adjacent to or within EphB4-expressing tissues. These findings indicate that EphB4 receptors may interact in vivo with multiple B-class ephrins. The expression patterns also suggest that EphB4 receptors and their ligands may be involved in visceral arch formation, somitogenesis, and blood vessel development.
REFERENCE COUNT: 69 THERE ARE 69 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 61 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:126378 CAPLUS
DOCUMENT NUMBER: 130:350145
TITLE: Roles of ephrinB ligands and EphB receptors in cardiovascular development: demarcation of arterial/venous domains, **vascular** morphogenesis, and sprouting angiogenesis
AUTHOR(S): Adams, Ralf H.; Wilkinson, George A.; Weiss, Cornelia; Diella, Francesca; Gale, Nicholas W.; Deutsch, Urban; Risau, Werner; Klein, Rudiger
CORPORATE SOURCE: European Molecular Biology Laboratory, Heidelberg, D-69117, Germany
SOURCE: Genes & Development (1999), 13(3), 295-306
CODEN: GEDEEP; ISSN: 0890-9369
PUBLISHER: Cold Spring Harbor Laboratory Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Eph receptor tyrosine kinases and their cell-surface-bound ligands, the ephrins, regulate axon guidance and bundling in the developing brain, control cell migration and adhesion, and help patterning the embryo. Here we report that two ephrinB ligands and three EphB receptors are expressed in and regulate the formation of the **vascular** network. Mice lacking ephrinB2 and a proportion of double mutants deficient in EphB2 and EphB3 receptor signaling die in utero before embryonic day 11.5 (E11.5) because of defects in the remodeling of the embryonic **vascular** system. Our phenotypic anal. suggests complex interactions and multiple functions of Eph receptors and ephrins in the embryonic **vasculature**. Interaction between ephrinB2 on arteries and its EphB receptors on veins suggests a role in defining boundaries between arterial and venous domains. Expression of ephrinB1 by arterial and venous endothelial cells and EphB3 by veins and some arteries indicates that endothelial cell-to-cell interactions between ephrins and Eph receptors are not restricted to the border between arteries and veins. Furthermore, expression of ephrinB2 and EphB2 in mesenchyme adjacent to vessels and **vascular** defects in ephB2/ephB3 double mutants indicate a requirement for ephrin-Eph signaling between endothelial cells and surrounding mesenchymal cells. Finally, ephrinB ligands induce capillary sprouting in vitro with a similar efficiency as angiopoietin-1 (Ang1) and **vascular** endothelial growth factor (VEGF), demonstrating a stimulatory role of ephrins in the remodeling of the developing **vascular** system.
REFERENCE COUNT: 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 62 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:295080 CAPLUS
DOCUMENT NUMBER: 130:323337
TITLE: Transmembrane ephrin ligands in neural and **vascular** development
AUTHOR(S): Wang, Hai
CORPORATE SOURCE: California Institute of Technology, Pasadena, CA, USA
SOURCE: (1998) 116 pp. Avail.: UMI, Order No. DA9912892
From: Diss. Abstr. Int., B 1999, 59(11), 5721
DOCUMENT TYPE: Dissertation
LANGUAGE: English
AB Unavailable

L8 ANSWER 63 OF 67 USPATFULL
ACCESSION NUMBER: 1998:39241 USPATFULL
TITLE: Cytokines that bind the cell surface receptor hek
INVENTOR(S): Beckmann, M. Patricia, Poulsbo, WA, United States
Cerretti, Douglas P., Seattle, WA, United States
PATENT ASSIGNEE(S): Immunex Corporation, Seattle, WA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5738844		19980414
APPLICATION INFO.:	US 1995-453943		19950530 (8)

RELATED APPLN. INFO.: Division of Ser. No. US 1994-240124, filed on 9 May 1994, now patented, Pat. No. US 5516658 which is a continuation-in-part of Ser. No. US 1993-161132, filed on 3 Dec 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-114426, filed on 30 Aug 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-109745, filed on 20 Aug 1993, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Ulm, John
ASSISTANT EXAMINER: Mertz, Prema
LEGAL REPRESENTATIVE: Anderson, Kathryn A.
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
LINE COUNT: 1718
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Hek ligand (hek-L) polypeptides as well as DNA sequences, vectors and transformed host cells useful in providing hek-L polypeptides. The hek-L polypeptides bind to a cell surface receptor (hek) that is a member of the receptor tyrosine kinase family. Hek is expressed on cells that include certain tumor cell lines. The hek-L polypeptides also bind a distinct receptor tyrosine kinase known elk.

L8 ANSWER 64 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1998:700539 CAPLUS
DOCUMENT NUMBER: 130:47629
TITLE: Cell-type specific and estrogen dependent expression of the receptor tyrosine kinase EphB4 and its ligand ephrin-B2 during mammary gland morphogenesis
AUTHOR(S): Nikolova, Zariana; Djonov, Valentin; Zuercher, Gisela; Andres, Anne-Catherine; Ziemiecki, Andrew
CORPORATE SOURCE: Department of Clinical Research, University of Berne, Bern, CH-3004, Switz.
SOURCE: Journal of Cell Science (1998), 111(18), 2741-2751
CODEN: JNCSAI; ISSN: 0021-9533
PUBLISHER: Company of Biologists Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Morphogenesis of the mammary gland occurs mainly during adult life and is dependent on a complex interplay of hormonal, cell-cell and cell-matrix interactions. The mol. mechanisms involved in pattern formation of the mammary epithelium in adult life are poorly understood. Recently, several members of the Eph family of receptor tyrosine kinases and their ligands have been shown to participate in pattern formation during embryogenesis and conceivably may fulfill similar functions during adult morphogenesis. The authors have investigated the expression of a member of this family, EphB4, and its cognate ligand, ephrin-B2, during normal and malignant mouse mammary morphogenesis. A spatially, temporarily and hormonally coordinated expression of both the receptor and ligand was obsd. The receptor was predominantly localized in the myoepithelial cells surrounding the ducts and alveoli, whereas ligand expression was limited to the luminal epithelial cells. Expression of both was induced at the onset of gland morphogenesis at puberty and was differentially regulated during the estrus cycle. Ovariectomy of prepubertal or adult females abolished the expression of both receptor and ligand and administration of estrogen alone was sufficient to restore their normal expression. Disruption of the balanced expression was obsd. during exptl. mouse mammary carcinogenesis. Ligand expression was lost at the onset of **tumorigenesis** and receptor expression shifted from myoepithelial to epithelial cells with progressive malignancy. These results implicate both the EphB4 receptor and its ligand ephrin-B2 in the hormone dependent morphogenesis of the mammary gland. Furthermore, their deregulated expression may contribute to mammary carcinogenesis.

REFERENCE COUNT: 61 THERE ARE 61 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 65 OF 67 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 3
ACCESSION NUMBER: 1996:350544 CAPLUS
DOCUMENT NUMBER: 125:50767
TITLE: Cytokines that bind cell surface receptor tyrosine kinase hek, **tumor** cells, oligonucleotide probes, recombinant expression, and therapeutic and diagnostic uses
INVENTOR(S): Beckmann, M. Patricia; Cerretti, Douglas P.
PATENT ASSIGNEE(S): Immunex Corp., USA
SOURCE: U.S., 19 pp., Cont.-in-part of U.S. Ser. No. 161,132, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5516658	A	19960514	US 1994-240124	19940509
CA 2169851	AA	19950302	CA 1994-2169851	19940817
WO 9506065	A1	19950302	WO 1994-US9282	19940817
W: AU, CA, FI, JP, KR, NO, NZ				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9475675	A1	19950321	AU 1994-75675	19940817
AU 680540	B2	19970731		
JP 09502174	T2	19970304	JP 1994-507650	19940817
EP 804482	A1	19971105	EP 1994-925915	19940817
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, NL, SE, MC, PT, IE				
US 5738844	A	19980414	US 1995-453943	19950530
FI 9600743	A	19960219	FI 1996-743	19960219
NO 9600652	A	19960219	NO 1996-652	19960219
US 5969110	A	19991019	US 1998-57121	19980408
US 6274117	B1	20010814	US 1999-358734	19990721
US 2002010325	A1	20020124	US 2001-904954	20010712

PRIORITY APPLN. INFO.:
US 1993-109745 B2 19930820
US 1993-114426 B2 19930830
US 1993-161132 B2 19931203
US 1994-240124 A 19940509
WO 1994-US9282 W 19940817
US 1995-453943 A3 19950530
US 1998-57121 A3 19980408
US 1999-358734 A3 19990721

AB Hek ligand (hek-L) polypeptides as well as DNA sequences, vectors and transformed host cells useful in providing hek-L polypeptides are studied.
The hek-L polypeptides bind to a cell surface receptor (hek) that is a member of the receptor tyrosine kinase family. Hek is expressed on cells that include certain **tumor** cell lines. The hek-L polypeptides also bind a distinct receptor tyrosine kinase known elk. Diagnostic and therapeutic applications of hek-L are recognized.

L8 ANSWER 66 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1995:632147 CAPLUS
DOCUMENT NUMBER: 123:31254
TITLE: Cytokine ligands of the cell surface receptor HEK and cDNAs encoding them
INVENTOR(S): Beckmann, M. Patricia; Cerretti, Douglas P.
PATENT ASSIGNEE(S): Immunex Corp., USA
SOURCE: PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9506065	A1	19950302	WO 1994-US9282	19940817
W: AU, CA, FI, JP, KR, NO, NZ				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5516658	A	19960514	US 1994-240124	19940509
AU 9475675	A1	19950321	AU 1994-75675	19940817
AU 680540	B2	19970731		
JP 09502174	T2	19970304	JP 1994-507650	19940817
EP 804482	A1	19971105	EP 1994-925915	19940817
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE				
FI 9600743	A	19960219	FI 1996-743	19960219
NO 9600652	A	19960219	NO 1996-652	19960219

PRIORITY APPLN. INFO.:
US 1993-109745 A 19930820
US 1993-114426 A 19930830
US 1993-161132 A 19931203
US 1994-240124 A 19940509
WO 1994-US9282 W 19940817

AB Proteins that bind the cell surface receptor HEK (HEK ligand or hek-L polypeptides) are characterized and DNA sequences encoding them are cloned for use in the manuf. of the ligands. The HEK cell surface receptor is a member of a receptor tyrosine kinase family that is found on a no. of normal and **tumor** cell lines. The hek-L polypeptides also bind a distinct receptor tyrosine kinase known elk. Cells synthesizing the ligand were identified by screening with fusion protein of HEK and an antibody. CDNAs for the ligands were cloned by screening a bank from the T-cell leukemia line CCRF-HSB-2 in pCD410 by screening the bank in CV1/EBNA-1 cells for the ligand using the fusion protein described above. The ligand bound to the fusion protein with a Ka of 2.times.108 M-1.

L8 ANSWER 67 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1995:498000 CAPLUS
DOCUMENT NUMBER: 122:287341
TITLE: Protein tyrosine kinases expressed in glomeruli and cultured glomerular cells: FLT-1 and VEGF expression in renal mesangial cells
AUTHOR(S): Takahashi, Takamune; Shirasawa, Takuji; Miyake, Keiko; Yahagi, Yuichi; Maruyama, Naoki; Kasahara, Naruhiko; Kawamura, Testsuya; Matsumura, Osamu; Mitarai, Tetsuya; Sakai, Osamu
CORPORATE SOURCE: Dep. Mol. Pathol., Tokyo Metropolitan Inst. Gerontol., Tokyo, 173, Japan
SOURCE: Biochemical and Biophysical Research Communications (1995), 209(1), 218-26
CODEN: BBRCA9; ISSN: 0006-291X
PUBLISHER: Academic
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Protein tyrosine kinases play an important role in cellular proliferation and differentiation of various cell types. To identify potential tyrosine kinases involved in glomerular functions the authors have utilized the PCR and degenerate oligonucleotides for isolation of such genes from isolated glomeruli, cultured mesangial cell, and glomerular endothelial cells. Sequence anal. of PCR-amplified cDNAs resulted in the isolation of 24 tyrosine kinases. Here the authors describe for the first time the constitutive expression of 15 tyrosine kinases, tyro-1, tyro-4, tyro-6, hyk, Ptk-3, Ryk, tie, yes, lyn, tec, Jak1, Jak2, Jak3, c-abl, and flk, in renal glomeruli. In addn., Flt-1, an endothelial cell-specific receptor for **vascular** endothelial growth factor (VEGF), is expressed in renal mesangial cells, and its gene expression is up-regulated upon the stimulation of platelet-derived growth factor (PDGF) with the concomitant up-regulation of VEGF. These data suggest the possible involvement of VEGF/Flt-1 system in cytokine-induced mesangial cell proliferation.